

THE [REDACTED]ARY OF PRACTICAL MEDICINE

PREFACE

LARGELY as the result of medical research and practice in connexion with the late War, much light has been shed on the nature of many diseases during the last few years. The knowledge thus acquired is, as far as the scope of the work has permitted, duly recorded in these pages.

The work is, as it claims to be, a Dictionary of *Practical Medicine*. Its appeal is chiefly to the practitioner, whose needs have been kept steadfastly in mind throughout. Etiology and Pathology have, of course, received due attention, but elaborate disquisitions on unsettled questions have as far as possible been avoided so that more space might be available for Symptomatology, Diagnosis, Prognosis, and Treatment. In brief, the governing aim of the work is to assist the practitioner in the recognition and treatment of disease.

The Dictionary covers the whole range of Medicine, including the chief tropical and industrial diseases, except that Labour, both normal and difficult, and Fractures and Dislocations are omitted as subjects too extensive for consideration in a work on General Medicine limited to three volumes. The Diseases of Pregnancy and the Puerperium and Diseases of Women in general are, however, included.

Descriptions of Major Operations have been excluded as lying outside the scope of the work, but the indications for them are given, and Minor Operations are described with needful detail.

The modern methods of Diagnosis and Treatment are expounded, and their significance and value assessed. Consideration is also given to such general subjects as Immunity, Anæsthetics, Clinical Bacteriology and Pathology, the modern developments of Chemical Pathology, the examination of candidates for Life Insurance, and the problems of Forensic Medicine.

As the originator of the work, I desire to pay ungrudging tribute to the labours of my co-Editors. Dr. Gordon Holmes has had charge of the Neurology, and I of the Dermatology, while Prof. Langmead shares with me the editorial responsibility for all the rest of the work.

They join with me in acknowledging the courtesy with which editorial requests and suggestions have been received by the contributors. While the general teaching of the work will be found to be harmonious, the Editors have

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not sought unduly to limit the discretion of individual writers on points of detail, preferring that on such points each should give the results of his own experience.

We desire also to thank contributors and others who have provided preparations, drawings, or photographs for the Plates and the Figures in the text, and especially Dr. Bigland, Sir John Broadbent, Dr. Cammidge, Dr. d'Este Emery, Dr. Dore, Dr. Golla, Dr. Gray, Col. Harrison, Dr. MacCormac, Dr. MacLeod, Dr. Manson-Bahr, Prof. Murray, Mr. Pannett, Dr. Parkinson, Mr. Magnus Redding, Dr. Rolleston, Dr. Sequeira, Dr. Gordon Thomson, and Mr. Thomson Walker.

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THE DICTIONARY OF PRACTICAL MEDICINE

ABDERHALDEN REACTION (*see* SEROLOGICAL DIAGNOSIS).

ABDOMINAL INJURIES. Anatomy.—

From the point of view of susceptibility of its contents to injury, the abdomen can be roughly divided into three parts: an upper zone lying under cover of the ribs, a lower portion protected by the bony ring of the true pelvis, and an intermediate zone reaching from the ribs to the pelvis. From the costal margin to the pelvic girdle stretch several muscle sheets (external oblique, internal oblique, transversalis, rectus) which serve as a strong shield against external violence; far back in the loin the vertical fibres of the erector spinæ and quadratus lumborum give protection.

Injury to the pelvic contents is almost always associated with fracture of the bony pelvis, of which we do not treat in this work; we shall confine ourselves to the parts more exposed to injury, i.e. the upper and intermediate zones of the abdomen.

The solid abdominal organs (liver, spleen, kidneys, and pancreas) are situated mainly above the level of the umbilicus; the hollow viscera (stomach, intestines, biliary and urinary channels) are distributed over the whole cavity. Since the crushing of the solid viscera is fraught with more immediate peril, it is of advantage that they are partially protected by the costal framework; the hollow viscera are more mobile, and can sometimes evade violence by slipping aside. Evasion of impending injury is thus possible for intestine supplied with mesentery, but if the strain falls on a part of the gut which is near a fixed portion and has limited mobility (e.g. the duodeno-jejunal junction), severe tearing may follow.

The aorta and vena cava inferior, with their branches and tributaries, lie far back in the abdomen, and are seldom injured without other viscera undergoing severe trauma; but the common mesentery of the small intestine

is occasionally torn, and dangerous bleeding may occur from the lacerated vessels.

Etiology.—Injury of the abdominal wall may be produced by any kind of violence projected against the abdomen.

Injury of the abdominal contents may be due to penetrating or non-penetrating violence. Penetrating wounds are inflicted by sharp (cutting or stabbing) implements, by bullets, or by severe blows from blunt objects such as the end of the shaft of a cart. Non-penetrating injuries are most commonly the result of severe crushes, such as would follow from a heavy vehicle running over the abdomen; in the upper zone the lower ribs are sometimes fractured and driven on to the subjacent viscera. Another type of violence is the sharp circumscribed blow due to a kick, punch, or the sudden impinging of any hard body against the abdominal wall. Severe injury may also follow from the strain on the visceral attachments consequent on a fall from a height, or a sudden trip-up causing a violent fall forward.

When violence is applied against the abdominal wall the kind of injury produced depends to a certain extent upon the preparedness of the patient for the blow and the consequent rigidity or flaccidity of the muscular layers. A rigid muscle may mitigate or prevent injury of the deeper-lying viscera.

Varieties of injury and symptomatology.

—For purposes of description abdominal injuries are best divided into—

1. Injuries of the abdominal wall.
2. Injuries of the abdominal viscera.
3. **Injuries of the abdominal wall** comprise:
 - i. Simple contusions.
 - ii. Incision or laceration of the superficial parts.
 - iii. Incision or laceration of the whole thickness of the wall—the penetrating injury.
 - iv. Rupture of the deeper parts of the abdominal wall without the skin being broken.

ABDOMINAL INJURIES

Let it be constantly remembered that *any injury to the abdominal wall, however trivial, may be accompanied by serious lesions of the viscera, and that the latter may be seriously damaged without any visible sign of injury to the abdominal wall.*

Injuries of the abdominal wall alone are not, as a rule, accompanied by severe shock, even though the muscles be torn. Simple contusions of the abdominal wall are usually accompanied by tenderness and rigidity over the bruised area, but if no other lesion be present the rigidity passes off within a few hours. The persistence of rigidity betokens something more serious. In connexion with small wounds of the abdominal wall made with sharp instruments, it must be emphasized that an apparently superficial wound is often in reality a penetrating one. Errors in diagnosis are sometimes made owing to the fact that when a probe is inserted the muscles contract and prevent its passage through the narrow slit made by the injuring weapon.

When the laceration or wound of the abdominal wall is considerable in extent, portions of omentum or intestine may prolapse. It is surprising how little shock and pain may accompany such evisceration.

If the deeper parts of the abdominal parietes be ruptured, the subjacent viscera may bulge into the gap and lie covered merely by skin and subcutaneous tissue; this condition is termed a traumatic ventral hernia, and occurs but rarely.

2. In considering injuries of the abdominal viscera the chief interest lies in those cases which present no wound of the abdominal wall. When such wound exists the possibility of injury of the viscera is always considered, and is decided by carefully exploring the wound to determine penetration or otherwise. When, however, there is no external wound or only a slight contusion, the thought of visceral injury is not so insistent, and early symptoms of grave lesions may pass unheeded.

The viscera may be contused or ruptured. It is frequently impossible to diagnose between these unless the abdomen be opened or until lapse of time decides, but it is possible to lay down some rules for guidance in treatment after considering the true value of the various symptoms which may develop.

The main symptoms following injury to the abdominal viscera are due to shock, internal hæmorrhage, peritonitis, or a combination of these.

Shock is shown by pallor, feeble pulse, slow shallow respiration, and cold extremities, but unless there is some serious lesion the symptoms soon subside. If the state of shock lasts longer and seems out of proportion to the evidence of intra-abdominal injury, examination may reveal a pneumothorax or other chest condition. Injuries of the upper abdomen cause more serious shock than those of the lower portion. Renal contusions also cause severe shock.

If the symptoms of shock do not pass off within six to twelve hours, hæmorrhage or peritonitis is almost certainly an additional factor. Speaking generally, rupture of solid organs leads to hæmorrhage, injury to hollow viscera to peritonitis.

Owing to the anatomical disposition of the parts, hæmorrhage usually follows lesions of the upper zone, in which lie the liver, spleen, and kidneys. The main abdominal blood-vessels are more likely to be injured by violence directed against the central portion of the abdomen. When the liver or spleen is very severely torn, symptoms of shock and hæmorrhage commingle, and death frequently follows soon after the injury. In lesser degrees of injury of the solid viscera, evidences of bleeding gradually assert themselves. Increasing restlessness and pain, progressive pallor of the lips and finger-nails, rising pulse-rate and movable dullness in the flanks are sufficiently indicative. Occasionally the symptoms of hæmorrhage may abate for a day or two, and then become alarmingly evident after some exertion, such as straining on the bed-pan. The pulse-rate is a good but not always strictly reliable guide in abdominal hæmorrhage; it may continue fairly slow till the abdomen is full of blood, and then suddenly bound up to a rapid rate.

Contusion or rupture of the *kidney* is frequently accompanied by very severe and alarming shock, which generally passes off within an hour or two (*cf.* the kidney-punch in boxing). The later symptoms depend on the extent of the injury and the condition of the renal capsule. In slight cases in which the capsule remains intact, hæmaturia, local tenderness, and possibly renal colic due to the passage of clots down the ureter comprise all the symptoms. If the renal capsule be torn a retroperitoneal hæmatoma is formed, and urine is extravasated into the cellular tissues behind and around the kidney; this leads to cellulitis, evidenced generally by malaise and

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irregular fever, and locally by swelling, tenderness, and muscular resistance. An abscess may result. If the urine be infected the symptoms are more acute. Should the peritoneal covering of the kidney be torn, symptoms of intraperitoneal hæmorrhage may result. Most cases of hæmaturia resulting from renal contusion stop spontaneously.

Peritonitis is usually consequent on rupture of the *hollow viscera*. The intestines, bladder, and stomach are most commonly injured; the gall-bladder and ureter rarely. Sometimes the injury is only sufficient to bruise the walls of the viscus. If the stomach is thus contused the result is vomiting, and sometimes hæmatemesis; if the colon is bruised the passage of blood per anum and diarrhœa due to traumatic colitis may follow. Bruising of the bladder causes slight hæmaturia, but if the contusion be very severe there may be vomiting and local rigidity even though no rupture has occurred.

Rupture of *intestine* is the commonest cause of peritonitis after abdominal injury; it is a condition fraught with danger of almost certain death if not diagnosed early, yet the signs and symptoms are often equivocal for some hours. Generally the tear only involves a portion of the circumference of the gut, but occasionally a complete severance is caused and a gap left between the two ends which may be temporarily closed by contraction of the involuntary intestinal muscle. When the intestine is injured its movements stop owing to a reflex or direct paresis of its walls. If the rent is small the edges of the mucous membrane pout and fill the small gap; a small amount of intestinal contents escapes and sets up a local plastic peritonitis with deposit of lymph which glues together the coils of intestine. The patient takes nothing by the mouth, and the intestines remain at rest. A lull in the symptoms gives a false security. After a few hours, when it is thought that there is no serious intra-abdominal lesion, food is taken, the intestines are excited to peristaltic contraction, and the opening in the gut is unsealed. Peritonitis then develops more or less rapidly according to the size of the opening and the number of adhesions. The important earlier signs of peritonitis are pain, local tenderness and muscular rigidity, vomiting, and shallow abdominal respiration. The later evidences of peritoneal infection are elevation of pulse and temperature, increasing distension, movable dullness in the flanks, and

obliteration or diminution of the liver dullness owing to free gas in front of the liver. The patient often has an anxious facial expression, and may show unusual restlessness. Rectal examination may demonstrate a tender pelvic peritoneum. In upper abdominal injuries, importance must be attached to a shifting of the pain to the hypogastrium. The last two symptoms depend upon the action of gravity in directing the inflammatory fluid and escaped intestinal contents into the lower abdomen and pelvis. The prognosis in cases of ruptured intestine is very bad unless diagnosis is made and operation undertaken soon after the injury. Hence the need for early diagnosis.

It is possible for the duodenum and part of the colon to be ruptured behind the peritoneum. The symptoms are then due to a retroperitoneal cellulitis, with some inflammation of the contiguous peritoneum, viz. local pain, muscular rigidity, shallow respiration, vomiting, and rise of pulse and temperature. A very important diagnostic sign, when present, is surgical emphysema of the retroperitoneal tissues.

Rupture of the *urinary bladder* usually occurs in connexion with fractured pelvis, but may result from a blow on the hypogastrium when the viscus is distended. In children the bladder is situated higher in the abdomen, and is therefore more liable to injury. The symptoms vary according as the rent is intra- or extraperitoneal. If it is within the peritoneum, symptoms of peritonitis ensue, but it must be recollected that sterile urine does not at first cause a very acute inflammatory reaction. There may be hæmaturia. Rupture outside the peritoneum leads to extravasation of urine, and consequent cellulitis in the suprapubic and perineal regions.

Rupture of the *stomach* quickly causes symptoms of general peritonitis; it is generally accompanied by some other lesion such as injury to the spleen or liver; a local collection of free gas near the stomach may yield a very tympanitic note on percussion.

The *pancreas* is seldom injured, and the symptoms of such injury are in no way distinctive.

While rupture of the pancreas is rare, epigastric injury is sometimes followed by a pseudo-pancreatic cyst in the lesser peritoneal sac.

Diagnosis of abdominal injuries.—If the practitioner has a clear view of the possible symptoms resulting from the different intra-abdominal injuries, he can in most cases make

ABDOMINAL INJURIES

a fairly accurate diagnosis. In wounds of the abdominal wall their exploration will reveal any internal lesion.

The essential point in diagnosis is to estimate the different proportions that shock, hæmorrhage, and peritonitis take in the production of the observed symptoms, and to judge from this the viscus injured and the nature of the lesion. It is frequently impossible to give a definite opinion for a few hours after the accident. The thorax must always be carefully examined to exclude pneumothorax and to detect any broken ribs. For the rest, each individual symptom must be given its true value in relationship to the others. Shock usually subsides within a few hours, and then hæmorrhage or peritonitis becomes increasingly evident. Pain, vomiting, local or general muscular rigidity, tenderness, alteration of pulse-rate, shallow respiration, diminution of liver dullness, free fluid in the abdomen—these are the main symptoms to note. By taking into consideration the part of the abdomen struck, it is possible in most cases to say which viscus is injured and what is the nature of the injury. In cases of suspected rupture of the bladder an additional means of diagnosis is present; the bladder is emptied by a catheter and a measured quantity of boric-acid lotion or saline is introduced and again drawn off. Any marked discrepancy between the amount introduced and that drawn off suggests rupture of the bladder.

Treatment.—If there is no accompanying intra-abdominal injury, wounds of the abdominal wall should be treated on general surgical principles. All stab wounds and any possible or certain penetrating wound must be enlarged and explored. Any prolapsed viscus must be cleansed with saline and replaced in the abdomen, a drain being left in the peritoneal cavity.

In cases in which injury to the viscera is suspected but no lesion of the abdominal wall is present, treatment is often doubtful for several hours, *but, provided that there is no chest injury and that renal trauma is excluded*, it is generally advisable to perform an exploratory cœliotomy in the following circumstances:

1. When severe abdominal pain persists for more than about six hours after an injury, if such pain be accompanied by either—

- (a) Vomiting,
- (b) Pulse gradually rising from the normal,
- (c) Persistent local rigidity tending to spread,

ABDOMINAL PAIN

(d) Deep local tenderness with shallow respiration.

2. When the pulse rises steadily and becomes weaker hour by hour, and the patient is very restless or shows increasing anæmia.

3. If the liver dullness is diminished or free fluid is found in the abdomen.

It is important not to give morphia until the question of operation has been decided.

When the initial symptoms of shock are so severe that the patient appears moribund, operative interference is hardly justifiable. Such cases are usually consequent on very severe injury to the viscera with great hæmorrhage, and operation may hasten the end. It is better to wait awhile to see if any recovery from the grave shock occurs, when operation may be undertaken with more justification.

Most cases of contusion of kidney may be treated expectantly, but if the hæmorrhage is excessive and the kidney pulped, nephrectomy may be essential. Perinephritic extravasation of urine must be treated by incision and drainage.

ZACHARY COPE.

ABDOMINAL PAIN, DIAGNOSIS OF.

In this article consideration is first given to *general* aspects of the subject, then to its *special* aspects (the characters, and associated conditions, of abdominal pain in individual diseases).

I. GENERAL ASPECTS OF ABDOMINAL PAIN

1. **The mechanism of pain production.**—The debatable question of "referred pains" will not be discussed. In connexion with it, however, it is important to bear in mind that the mode of production of abdominal pain may well be different in different disease processes. Certainly, no one of the hypotheses at present advanced affords an adequate explanation of all the forms of pain with which the practitioner is met. In some instances abdominal pain seems to be a direct and simple expression of morbid changes, inflammatory or other, in a diseased organ, through its intrinsic nerve supply. In others, pain is probably not expressed directly, but indirectly through a "reflex arc" in which afferent sensory impulses from the diseased organ to a segment of the central nervous system, and efferent sensory impulses to a point remote, it may be, from the organ itself, are concerned. "Referred pains" are thus explicable. In still other cases pain seems to have a direct vascular origin, due perhaps to changes in the calibre of the

ABDOMINAL PAIN, DIAGNOSIS OF

arterioles of the part in which the pain is felt.

2. **Sensations closely allied to pain.**—Such sensations as repletion, sinking, fullness, hunger, fluttering, throbbing, though not necessarily painful, often have important bearings upon the pain sense, combining with it, and adding differentia that are helpful in diagnosis. Thus, "hunger-pain" is a very definite composite sensation of this sort, associated often with peptic ulcer at the pylorus or in the duodenum. In neurotic patients these allied pain sensations are sometimes very elaborate and, in consequence, unhelpful diagnostically, unless it be by way of confirming the presence of neurosis.

3. **Intensity of abdominal pain.**—This is as difficult of estimation as is pain in other parts of the body; indeed, it is even more difficult, because of the more complex structures involved and because of the frequency of associated sensations not in themselves purely painful. The temperament, sex, age and general physique of the patient; the effect upon the facies, pulse, skin, musculature, and nutrition; the kind and amount of whatever therapeutic measures are found to bring relief—these are the criteria that must be employed, together with a large clinical experience, in order to assess the degree of pain which a patient is suffering.

4. **Investigation of pain relations.**—Some such scheme as the following should be carried out in every case presenting abdominal pain as its chief, or as an important, symptom. Such a scheme, carefully followed, yields much more exact evidence as to cause than does inquiry which lacks method. An effort should be made to confine the patient to the actual question, and care should be exercised to ensure that he understands it. So far as is possible, leading questions should be avoided.

i. **Situation.**—This includes not merely the situation in general, but the point of origin, the direction of spread (if any), the point of maximum intensity, and the points of most remote reference. (See Sect. 6.)

ii. **Time relations.**—Questions under this head include the time of day or of night, the abruptness or otherwise of onset, whether the pain increases steadily to a maximum, crescendo fashion, or is at its height immediately or soon after it begins, whether it is paroxysmal or continuous, or both.

iii. **Characters.** The patient should be asked to state in his own terms "what the pain is

like"—sharp, cutting, gnawing, shooting, throbbing, dragging, dull, heavy, colicky, gripping; these are terms that help the inquirer; such expressions as agonizing, excruciating, unbearable, are often only expressions of the patient's intolerance of pain in general.

iv. **Associated sensations.**—See Sect. 2.

v. **Associated effects.**—These are important. Does vomiting occur *as the result of the pain*? Or defæcation? Or micturition? Or sweating? Or pallor? Or flushing? Or increased or diminished pulse-rate? Or shallow breathing? (See also Sect. 3.)

vi. **Relation to taking food.** Is the pain related to meals? If so, how soon after a meal does it commence? If the pain is stated to begin just *before* a meal, the fact is of the same significance as though it is stated to begin three or four hours after a meal.

vii. **Determining factors.**—Is the pain brought on by exercise, or by any posture (including recumbency), by jolting, by emotional disturbance, by fatigue, by urination, by defæcation, by cough?

viii. **Methods of obtaining relief.**—Inquiry in this direction yields valuable data. Food may give relief, or starvation, or vomiting, defæcation, the eructation of gas or its passage per anum. Certain postures may ameliorate the pain, whether postures of the whole body or of certain parts. Pressure upon the painful area may cause relief. The effect of local applications, whether hot or cold, should be considered. Lastly, the result of drugs, such as bicarbonate of soda, alcohol, bromides, belladonna, and opiates.

5. **Tenderness.**—Pain signifies spontaneous discomfort complained of by the patient independently of examination; *tenderness* is discomfort elicited by the examining hand. These two symptoms should be investigated separately, and the notions should be kept apart, although they often fuse in actual cases. Thus, when pain is very severe, as in general peritonitis, palpation of the abdomen may seem to the patient merely to intensify the pain rather than to discover tenderness. Again, patients sometimes complain that they are "tender" or "sore," even when no examination is being conducted. But in the majority of instances the patient can distinguish quite plainly between pain and tenderness as defined above, and the distinction is then very useful. *Hyperæsthesia* is a form of skin tenderness or sensitiveness which is another useful element in diagnosis.

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6. **The seat of pain.**—This may be *general* or *local*. Even when general there is often an area of maximum local reference of the pain, important to make out if present. Abdominal pain is sometimes local at first, becoming general later. Or, starting as a general pain, it may become local. Both of these sequences carry at times valuable information. For convenience of description, local abdominal pain is spoken of as being *epigastric*, *hypochondriac* (right and left), *umbilical*, *hypogastric*, and *iliac* (right and left).

II. SPECIAL ASPECTS OF ABDOMINAL PAIN

The commonest causes of abdominal pain are the comparatively trivial forms of dyspepsia, gastric and intestinal. These do not call for detailed consideration here. More important causes are provided by certain thoracic diseases, and diseases arising within the abdominal cavity itself.

Thoracic diseases.—There are three diseases in particular in which the lesion is situated above the diaphragm, yet the pain may be entirely abdominal:

1. **Angina pectoris.**—The pain has sometimes an abdominal reference. Most often, in such cases, the pain is epigastric. It observes the usual features seen in anginal pain in general; it follows physical effort, is accompanied by sensations of distress or impending disaster, has associated vaso-motor disturbances, and is relieved by nitrites. Lesser degrees of heart pain may also be referred to the upper part of the belly, as in aortic regurgitation.

2. **Pericarditis.**—Here, again, if the pain incident to the disease is abdominal in its reference, the epigastrium and region of the xiphisternum are the most common sites. But the whole of the abdomen may be affected, so that the simulation of peritonitis, especially as the patient's facies and general state often betoken an acute illness, may be very close. The pain may be very severe. It is commonly affected by posture, being increased by the supine position and relieved by sitting or bending forward. Pressure, even if light, often increases it. Severe epigastric pain in an acute illness should always lead to careful auscultation over the heart, and especially at the base, for pericardial friction. There is usually a good deal of abdominal tenderness on palpation, and some muscular rigidity.

3. **Pleurisy, with or without pneumonia.**—This is the commonest thoracic disease to cause abdominal pain. Its site is frequently

the epigastrium, but it may be in either hypochondrium, or it may be generalized, affecting the whole abdomen, thus, as in pericarditis, involving a differential diagnosis from peritonitis. When it is remembered that an acute pneumococcic infection may lead to pneumonia and to peritonitis concurrently, it is seen that great difficulty may arise. This is so especially in the case of children. Both in pericarditis and in pleurisy the attendant cough usually leads to exacerbation of the abdominal pain when this is present.

Some other thoracic conditions call for brief mention in this connexion. *Emphysema* sometimes produces a dull aching pain in the upper belly. *Cough*, in influenza and in other catarrhal states, may lead to a good deal of abdominal pain, no doubt due to the strain imposed upon the muscles.

Diseases of the peritoneum. 1. **Acute general peritonitis.** Abdominal pain is an almost constant symptom; indeed, if we except certain states in which the patient's mental condition is clouded and (or) in which he is moribund, it may confidently be asserted that pain is invariable. The pain of acute peritonitis bears the same relation to the pains associated with other abdominal lesions that the headache of acute meningitis bears to the headache of other lesions in the brain. Both are the severest of their kind, and the most continuous. Peritoneal pain is increased by any sort of movement, whether of the patient's body and limbs, or by such acts as coughing, sneezing, micturition, and defecation. Even breathing is inhibited as much as possible in order to avoid increase of the pain by respiratory movements. The least pressure upon the abdomen is intolerable, and, if the observer succeeds in exerting this even a little, the releasing of the hand causes an acute exacerbation of the pain. The tenderness elicited by the examining hand is exquisite. There is usually great muscle rigidity, in most cases general over the belly. In cases in which the peritonitis begins locally the pain is apt to be largely concentrated at that spot, but it is seldom entirely so localized; and whether it be more acute there or not at first, it quickly becomes more general. The region of the navel is often an area of maximum severity if any one area is worse than another, which it seldom is.

In the cases of "primary" peritonitis (e.g. pneumococcus infection) the pain is not usually so abrupt in its onset as it is in the "perforative" cases, and this feature is of some help

ABDOMINAL PAIN, DIAGNOSIS OF

in differential diagnosis as between the two conditions.

2. **Acute local peritonitis.**—The pain and tenderness in this condition have, for the most part, the same features as are detailed under acute general peritonitis, but they are generally less urgent. They are more or less localized to the region where the diseased organ causing the peritonitic extension of inflammation is placed. But it must be remembered that every focal peritonitis tends to become general. (The common causes of acute local peritonitis are inflammatory lesions of the appendix, Fallopian tubes, uterus, stomach, duodenum, liver, gall-bladder, and spleen.)

3. **Subphrenic abscess** is the result of acute inflammation of the peritoneum below the diaphragm and also of the lesser sac. Pain is almost always present in this condition, is generally referred to the right hypochondrium (but sometimes to the left), and is often also severe in the right flank and in the back. Tenderness, too, is exceedingly common, and is elicited in the same situations.

4. **Chronic general peritonitis.**—The most common cause is tuberculosis. Disseminate new growth, chronic mixed nephritis, and cirrhosis of the liver are less frequent causes.

In *tuberculous peritonitis* the degree of pain is extraordinarily variable. In some of the "plastic" cases in children, and, though less frequently, in some of the "ascitic" cases also, pain may be quite absent. When it is present it is rarely severe, is not specially liable to affect any particular region, and is complicated by feelings of general discomfort and of distension. Colic is not uncommon. Tenderness is usually present, even if pain is not, but this again is very variable in degree and position.

Diseases of the stomach and duodenum.

—The seat of pain is chiefly the epigastrium and the left hypochondrium; but the zone of reference often includes the lower ribs and interspaces on the left side as high as the axilla. Posteriorly, it may involve the left scapular region.

1. In **gastritis** the severity of the pain depends upon whether the condition is acute or chronic, and also upon its cause. It is usually felt soon after taking food, is prone to be widely distributed, and is often associated with a sense of fullness. It is generally relieved by vomiting, and the relief may be complete. It is also relieved by starvation, but not, as a rule, by taking alkalis, nor by recumbency.

Some degree of tenderness is usually present in the region of the pain when the abdomen is palpated. In acute gastritis the tenderness may be quite pronounced, and there may be some rigidity of the upper recti.

2. In **hyperchlorhydria** the pain is more localized in most instances than in gastritis, is relieved by vomiting, but, far from being relieved by starvation, is generally lessened by the taking of food, especially if this be in fluid form. If the pain has a crescendo character, rising to a maximum about an hour and a half after a meal, this feature is strongly suggestive of hyperchlorhydria. The pain is also relieved by bicarbonate of soda. As a rule, there is no tenderness in abdominal examination.

3. In **gastric ulcer** the pain has the same features as in hyperchlorhydria. Pain does not, of itself, enable the observer to distinguish between acid gastric dyspepsia and gastric ulcer. The pain may certainly be quite as severe in the former as in the latter condition. If, however, it be more or less constant over a long period, this fact favours hyperchlorhydria; whereas the occurrence of intermissions, lasting for some days or weeks, favours ulceration.

4. In **duodenal ulcer** the pain, which is apt to be very severe, follows a meal after a longer interval than in ulcer of the stomach or in hyperchlorhydria, and is often very regular in its appearance: 11 A.M., 5 P.M., 11 P.M., and 5 A.M. are very common times. It is not infrequently associated with an uncomfortable sense of hunger—"hunger-pain." The situation of the pain is, in the majority of cases, the right hypochondrium, immediately below the costal arch, but it may be beneath the lower right ribs themselves, and in some instances it is in the epigastrium; rarely it is in the left hypochondrium. As in gastric ulcer, food and bicarbonate of soda relieve the pain, though not so quickly.

The pain of peptic ulcer, whether gastric or duodenal, is relieved by movement rather than by rest; pressure upon the upper part of the belly may also allay it.

Contrary to what is often taught, abdominal palpation does not often elicit points of local tenderness in uncomplicated cases of peptic ulcer. If perigastritis or periduodenitis be present, however, local tenderness, with or without muscular rigidity, is common.

5. In **cancer of the stomach** the relation of the pain to meals is usually not nearly so dis-

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tinct as in peptic ulcer, is not so localized, nor is it often relieved by taking more food, or so completely by alkalis. The pain often "goes through to the back," and when this is the case the point of reference is generally below the level of the scapulæ, and more mesial than in simple ulcer or gastritis. Vomiting (a more constant feature than in ulcer and gastritis) brings little or no relief. It is important to note, however, that in some cases of gastric cancer the pain has all the features described in simple ulcer. On abdominal palpation, tenderness is much more common here than in simple ulcer, and if a palpable tumour be present this is almost invariably tender, and sometimes very much so.

6. In dilatation of the stomach, if pain be present, it is usually not severe, and is of a dull aching or dragging kind. It is often increased by assuming the erect posture, and is in most cases relieved by recumbency, and also, it may be completely, by vomiting. It is generally situated well to the left of the mesial line, even as far outwards as the flank. It is apt to be more severe when the dilatation is secondary to the pyloric obstruction than when it is due to muscular atonicity. Tenderness on palpation of the abdomen is usually absent.

Diseases of the gall-bladder and bile-ducts. 1. **Gall-stones.**—The engagement of a gall-stone in one of the bile-ducts leads to "biliary colic," a very severe form of paroxysmal pain. The onset of the attack is abrupt. It may follow some sudden effort, or jolting, or vibration; or it may arise without any such determining factor. The pain is at first usually referred to the right hypochondrium, or to the region of the right costal arch. In relatively few cases it is referred to the epigastrium, and in still fewer to the left hypochondrium. The pain, which quickly attains its maximum, radiates across the upper abdomen, through to the back and upwards in the direction of the right scapula and shoulder; it is often associated with vomiting, with a shiver, with a sense of flatulence, and with sweating. It is rarely relieved by any less powerful measure, whether local or general, than a hypodermic injection of morphia.

2. **Acute cholecystitis.**—Acute inflammation of the gall-bladder, whether associated with gall-stones or not, leads most often to severe pain in the right hypochondrium. It is more or less continuous, with acute exacerbations, some of which may be distinctly colicky in

nature. The radiation of the pain may follow that in gall-stone colic (*see above*). Palpation of the abdomen nearly always elicits great tenderness, superficial and deep, in the right hypochondrium, with some muscle rigidity. When by careful palpation the rigid rectus is overcome, a tender enlarged gall-bladder may often be felt, though this will depend much upon the freedom of the organ to enlarge and its ability to become swollen by increase of contained mucus, etc. Fever and the symptoms of an acute infection are common accompaniments of the pain.

3. **Chronic cholecystitis** yields pain of a similar kind, but much less severe. There is much less local tenderness, and the signs of infection are much less apparent.

Diseases of the pancreas. 1. **Acute hæmorrhagic pancreatitis.**—In this disease the pain is very abrupt in onset—the patient having been quite well at the moment of its first incidence—is very severe, and is a conspicuous element in the case. It is continuous, with colicky exacerbations. Although in some cases the upper zone of the belly is chiefly affected at first, the whole abdomen is often involved after a few hours. The upper zone is very tender, and often the tenderness quickly becomes general. Great muscular rigidity is present, again chiefly in the upper zone, at least at first. Vomiting occurs almost invariably, but brings no relief to the pain, and is followed by more or less persistent retching after the stomach is empty. The patient's facies is much affected, and the general state soon becomes one of collapse. Fever is not a feature. The diagnosis is (a) from a peritonitis of the "perforative" kind, especially following the escape of gastric or duodenal contents through a "peptic" ulcer, (b) from intestinal obstruction, and (c) from acute poisoning. The absence of any abatement of the pain and muscular rigidity with time—should it be decided to wait—usually makes laparotomy urgent, and the real cause of the symptoms is made clear. A correct diagnosis before the abdomen is opened must at present be regarded as largely in the nature of a happy guess, although a clear history of previous illnesses attributable to cholelithiasis, the patient being an adult, and the absence of any cause of ptomaine or chemical poisoning, may perhaps justify the hazard.

2. In **chronic pancreatitis** there is nothing in the characters of the abdominal pain which may be present to distinguish the condition

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from chronic gastritis, gastric cancer, or malignant disease of the head of the pancreas.

3. **Pancreatic calculus**, a rare disease, leads to "pancreatic colic," which is seldom distinguishable in its general features from biliary colic. It is said that the maximum intensity of the pain is situated to the left of the mesial line, but this may be the case in cholelithiasis (q.v.), a very much commoner condition. Fatty stools do no more than suggest the possibility of a colic being pancreatic. The discovery of a pancreatic stone in them, however, following an attack, may be regarded as justifying a diagnosis.

Diseases of the intestines. 1. **Duodenal ulcer.**—This has been referred to in connexion with the stomach.

2. **Enteritis**, whether acute or chronic, leads to general abdominal pain, which is apt to be colicky at times. The reference is often made rather specially to the region of the umbilicus. The pain is relieved by defæcation, by local heat, and sometimes by pressure.

3. **Colitis.**—Here the colicky nature of the pain is generally more distinct, and in acute colitis there may be tenesmus. The pain in general colon disturbances is not infrequently referred to the hypogastric region.

4. **Appendicitis.**—In acute appendicitis pain is probably a constant feature. It is usually the initial symptom, is generally severe, and is most often referred to the right lower quadrant in the first instance. As the disease progresses the pain may become more general, or it may be most pronounced in the epigastric and umbilical regions, or even in the left lower quadrant. Not infrequently it becomes much less severe, or may even depart altogether, when the associated peritonitis and severe constitutional symptoms become definitely established. Exacerbations of the pain, and sometimes definite colic, may complicate the condition. In chronic appendix disease pain is not a constant feature, and, when present, is often referred to the epigastrium, in association with gastric symptoms. There is a true *appendix colic*—attacks of paroxysmal abdominal pain occurring in connexion with an appendix which is either in a state of partial obliteration as regards its lumen or is adherent to neighbouring structures, and most often to the colon.

5. **Focal lesions of the colon**, such as new growth, give rise to pain of a fairly severe character, situated in the region of the lesion, but also in many cases complicated by the pain of chronic intestinal obstruction, due to

distension of the gut behind the block and ineffectual peristalsis.

6. **Acute intestinal obstruction.**—The pain is intense, persistent and urgent. It is usually fairly general in situation, seldom giving much guide to the site of the mechanical difficulty. The pain is not relieved by vomiting.

7. **Enteroptosis and intestinal stasis.**—Pain in these conditions is more often in the nature of chronic discomfort, being associated in the patient's mind with sensations of fullness, repletion, dragging, etc.

Bladder and prostate diseases.—Bladder pain is not often severe in respect of its abdominal reference; it is usually a dull aching pain just above the pubes. Associated deep-seated pelvic pain and perineal pain are common, and these are the rule if prostatic lesions are the chief cause of the pain. The zone of reference of prostatic pain and discomfort is prone to be more considerable than is commonly known; this is especially so when there is malignant disease of this organ; the whole of the lower abdomen may then be affected, together with the hips, sacrum, groins, and thighs.

Diseases of the kidney.—The pain of renal diseases is not, strictly speaking, abdominal in most instances. But mention may well be made of it here, seeing that it not seldom calls for differential diagnosis. 1. **Renal colic**, in its classical form, does not usually present difficulty. But aberrant forms may confuse with biliary colic, with pyloric spasm, with enterospasm, and with appendicular colic. Careful urinary and radiographic investigations provide the only data upon which a positive conclusion can be based. 2. **Dietl's crises**, occurring in nephroptosis, are probably rarer than most accounts suggest. The features of an attack are in themselves almost indistinguishable from a renal colic. The patient is a woman, the pain is usually right-sided, and there are clear signs of nephroptosis. 3. **Ureteric calculus** sometimes causes fairly severe pain in the iliac region, especially if the stone becomes impacted. The reference is usually to the groin, as in acute renal pain. The diagnosis is from appendix pain chiefly, when on the right side.

Vertebral disease, and especially Pott's disease, may cause pain with an abdominal reference. The zone is usually the upper, and the site of maximum pain is the epigastrium, and thus the pain of peptic ulcer is simulated, or, even more closely at times owing to its

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greater constancy, the pain of abdominal aneurysm. *The spine should always be carefully examined in a case of obscure abdominal pain.*

Tabetic crises.—The most common form is the so-called "gastric crisis." The importance of this condition largely turns upon the fact that it may arise very early in the course of tabes, whilst as yet there is little in the way of physical signs. The onset is sudden; pain, chiefly epigastric, is very acute, and usually spreads upwards and to the back. Vomiting is almost invariable, and may persist long after the stomach is empty. The tongue is sometimes strikingly clean. The patient may show symptoms of collapse. To remember tabes dorsalis as a cause of sudden attacks of pain and vomiting is to guard against an error in diagnosis.

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ABDOMINAL TUMOURS, DIAGNOSIS OF. Examination of the abdomen. It is important to make the inquiry into symptoms and history as full as possible. It is also important to investigate the gastric, intestinal, biliary, renal, and other functions, this part of the general examination being extended in the direction of the function of any organ which may seem to be associated with the tumour.

In regard to the abdominal examination proper, the practitioner should remember that, as *palpation* is by far the most helpful of all the methods used in the investigation of an abdominal swelling, care must be taken to ensure that the maximum data are obtained as the result of it. This means that the patient's posture must be such that the abdominal muscles are completely relaxed, also that the examining hands are warmed by immersion for five minutes in warm water, and that the patient's confidence is gained by gentle handling and the withdrawal of inhibition by tactful conversation. It should also be borne in mind that the pads of the fingers feel more accurately than the tips, that the bimanual method should be employed whenever possible, and that palpation in the genu-pectoral position may yield important information. It is sometimes advisable to repeat a careful palpation after one or two enemata have been given to clear the colon, and before a meal, when the stomach is relatively empty.

Next to palpation, *inspection* (which naturally precedes it in the order of examination) is of most service. For this purpose a good and even light is essential, also a view of the

abdomen from the head and foot of the bed as well as from the side.

Both inspection and palpation are undertaken first with the abdomen at rest, then during deep inspiration. Inspection should also, in certain cases (e.g. enteroptosis), be conducted when the patient is standing.

Percussion and *auscultation*, though they yield far less information of value, should by no means be neglected.

Exceptional methods of examination are: palpation in a warm bath, with a view to overcoming muscle rigidity; palpation under general anaesthesia; examination by inspection and palpation after inflating the stomach or colon by water or by gas; X-ray screening after injection of oxygen into the peritoneal cavity. Neither of these methods is of very general use.

During the examination it is very helpful for the observer to dictate a note of his findings in regard to the tumour: its *situation*, *size*, *shape*, *consistency*, *mobility* (with respiration and by the examining hands), and whether or not, and to what degree, it is *tender*. With regard to *situation*, it is well to specify the apparent depth of the tumour from the surface, in addition to the anatomical region which it occupies. As to *size and depth*, it should be remembered that the beginner usually underestimates both of these things.

Material to be considered.—Although the term "abdominal tumour" is usually confined to masses or lumps arising, and lying, within the abdominal cavity, there are two other groups of swellings which call for detailed mention, seeing that they are found in the course of routine abdominal examination and require differential diagnosis. The first of these groups includes tumours of the abdominal wall, and in this group may be placed swellings which, though arising within the abdomen, protrude outside it, such as herniae. The second group includes tumours arising behind the peritoneum, among which the most common are tumours of the kidneys and of the suprarenal glands. After dealing with these two groups, consideration will be given to certain abdominal swellings which in reality consist of relatively normal structures that, for special reasons, behave like tumours. Then we shall discuss morbid conditions of intra-abdominal organs or tissues, leading to the formation of tumours, i.e. to abdominal tumours proper. Finally, multiple tumours will be very briefly considered.

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1. **Tumours of the abdominal wall.**—It is not always easy to decide if a tumour is situated in the parietes or within the abdominal cavity. The decision, however, is often a matter of considerable importance. Most experienced observers agree that, in this respect, tumours usually tend to lie more deeply than is thought, so that, in any doubtful instance, the probability is in favour of an intra-abdominal origin of the swelling.

Tumours of the abdominal wall are: neoplasm (sarcoma, carcinoma, lipoma, fibroma); abscess; actinomycosis; glanders; gumma.

Of the **neoplasms**, those presenting least difficulty are perhaps *lipomata*; they are often multiple, with irregular distribution on the trunk and limbs, the patient is often stout, there are no constitutional symptoms, and the "feel" of the mass is highly suggestive of its nature. *Sarcoma* is nearly always single; the patient is usually a child or young adult, the tumour is more uniform in its consistency, is not so freely movable, may be vascular, and there may be general symptoms (anaemia, loss of weight, some pyrexia). *Carcinomatosis*, as the name suggests, gives multiple swellings, often very widely distributed (scalp, wall of thorax, bones); they are small, involve the skin rather than the deeper parts of the parietes, may be dimpled at the centre, and may vary in size from week to week. The patient is usually known to have a primary growth elsewhere, and not infrequently this has been dealt with surgically some time before the appearance of the metastases, e.g. excision of mamma for cancer.

Abscess is diagnosed by the presence of more or less fluctuation, pain, tenderness, redness, pyrexia, leucocytosis, and the positive result of puncture. **Actinomycosis** in its early stages resembles sarcoma and gumma somewhat closely, but when suppuration occurs (usually the result of secondary infection by staphylococcus) the differential diagnosis is from simple abscess. Actinomycosis should always be suspected if the tumour is situated in the right lower quadrant of the abdomen, because there is a tendency for streptothrix infection of the appendix and caecum to involve the abdominal wall secondarily. Lack of uniformity in the swelling caused by a supposed abscess in the abdominal wall, whether as regards consistency or redness, should also suggest actinomycosis.

Gumma is not so common in the abdominal wall as in the thorax because it is most often

of periosteal origin. The same comment will apply to *tuberculoma* with suppuration. The presence of a positive Wassermann reaction, the absence of signs of suppuration, and good effects following antisyphilitic treatment are the chief points in establishing the diagnosis of gumma.

2. **Retroperitoneal tumours.**—Tumours consisting of the *kidneys*—whether displaced or enlarged by disease, or both—and of the *suprarenals* may be more conveniently considered later (Sect. 3, i, p. 12; Sect. 4, ii, p. 13).

Neoplasms arising in the retroperitoneal glands produce tumours on abdominal examination which not infrequently give rise to considerable difficulty in diagnosis. They may be primary, when they are usually sarcomatous in nature; or they may (less often) be secondary carcinomata, the primary disease being in the stomach or colon. In the former case the first symptoms are referable to the retroperitoneal mass; in the latter case that is rarely so. The condition is usually associated with a good deal of pain, which may be localized in the back, and a sense of considerable distension. The mass is apt to show the first signs of its presence in the upper zone of the abdomen and towards the mesial line, but it is of great importance to remember that *no mass as such may be felt, but various viscera*, and especially the liver and stomach, *may be thrust forward* so as to become unduly prominent and suggest that they themselves are enlarged or diseased. It was pointed out many years ago that areas of dullness to percussion, occupying unusual situations, and in the immediate neighbourhood of areas of normal or increased tympanitic resonance, suggest the presence of retroperitoneal tumour.

Psoas abscess is an important swelling arising behind the peritoneum. In the majority of cases it is secondary to Pott's disease, but it may follow perinephric suppuration. It usually leads to a tumour which is discovered in one or other lower quadrant of the abdomen—a fluctuating swelling, tending to spread downwards beneath Poupart's ligament, often free from tenderness, and not infrequently leading to spontaneous flexion of the thigh, the correction of which posture renders the swelling more prominent and perhaps painful. **Iliac abscess** lies farther from the mesial line, in the iliac fossa, and is much more restricted in regard to its situation. **Perinephric abscess**, though it may sometimes be felt as an ill-defined swelling in one or other lumbar region,

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usually gives signs in the loin—fullness on inspection and on bimanual palpation, and perhaps œdema of the skin. **Hæmatomata** may form behind the peritoneum. They are of more acute origin than abscesses, and are usually much more painful. They follow trauma, or operations, or they complicate hæmorrhagic diseases.

3. Tumours consisting of relatively healthy structures rendered more easily palpable than normal by some anatomical or physiological change.

i. The **kidney** is the most important of such tumours. There is no absolute criterion of the degree to which healthy kidneys are palpable. To feel the lower pole of the right kidney in a tall and thin woman during inspiration may be regarded as the rule rather than the exception. All degrees of nephroptosis may be found, up to complete displacement of the organ into the lower part of the abdomen or even across the mesial line. The right kidney is much more subject to such excursions than is the left. The diagnosis turns upon the shape and "feel" of the tumour, its sensitiveness, its being palpable from the loin (unless displaced entirely away from the flank), and its reducibility to its proper position by manipulation. In a doubtful case, in which the mass lies well away from the costal arch, diagnosis is, of course, considerably helped by the observer's inability to discover the kidney in its proper situation.

ii. The **liver** may be dropped as part of a general enteroptosis, giving the impression of a general enlargement. Or there may be undue prominence and elongation downwards of part of the right lobe (Riedel's lobe: "corset liver"). Both of these conditions are more common in females than in males.

iii. The **spleen** may be palpable in a general enteroptosis. Or it may be found as an isolated dropped organ, having got free from its supporting ligament. In the latter case it is not seldom enlarged, perhaps owing to partial obstruction to its vessels.

iv. The **abdominal aorta** is occasionally felt with such ease that it may be mistaken for an abnormal structure. The patient is usually a thin and nervous woman. The condition is not infrequently regarded by the beginner as an abdominal aneurysm. One or other of the **common iliac arteries** sometimes gives rise to a similar error.

v. The **lumbar spine**, if unduly prominent in a thin subject, may be unusually conspicuous, and may lead to difficulty in diagnosis.

vi. The **pylorus** may sometimes be felt, quite apart from being hypertrophied (as in hypertrophic stenosis), as a rounded insensitive structure, lying to the right of, or in, the mesial line, and rather nearer to the umbilicus than to the sterno-xiphoid junction. It is generally movable.

vii. The **descending colon** is quite commonly palpable in the left lumbar and left iliac region, especially if it happen to be somewhat contracted. It is felt as an elongated, rounded, almost insensitive structure, freely movable, and can be rolled under the hand.

viii. The **ascending colon** can be felt much less often, and (ix) the **stomach** but rarely.

x. The **distended urinary bladder** and (xi) the **pregnant uterus** may perhaps be included, not inappropriately, in this group of swellings. Both are frequently mistaken for morbid swellings, and it is of great importance to bear them constantly in mind. They both occupy the hypogastrium, are insensitive, elastic, and uniform in character. Confirmation of the one is yielded by the passage of a catheter into the bladder, and of the other by the presence of other signs of pregnancy.

4. **Abdominal tumours proper.** For convenience these will be dealt with under the various abdominal "regions" which they most often occupy. Either because the particular organ affected is displaced, or because it is considerably enlarged, a tumour involving any viscus may come to lie in some other region than the one to which it anatomically belongs. This fact, which must always be remembered, makes any classification based upon anatomical lines inadequate.

i. The **epigastrium**. The tumours in this region are most often of gastric origin, and *cancer of the stomach* is the most common. The case with which a gastric carcinoma can be felt depends upon its situation in the stomach, its size, and the position of the organ. Cancer of the lesser curvature close to the cardia, especially if its direction of spread be backwards, often escapes detection altogether by physical examination. In palpating for a suspected growth of the stomach, the regions of the epigastrium and left hypochondrium *close under the costal arch* should be critically investigated during deep inspiration. The tumour is usually tender, firm and irregular in form. *Cancer of the pylorus* leads to much less fixation of the organ than does cancer of the body, and for this reason the tumour may be

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felt in the umbilical region, and it may be movable.

Enlargements of the *liver* may be more conspicuous in the epigastrium than in the hypochondria when the mesial aspect of either lobe is chiefly involved. Not seldom a mass thought to be gastric cancer is found, during operation or at autopsy, to be formed chiefly by direct infiltration of the liver, to which the primary growth in the stomach is adherent.

Tumours, cysts, and inflammatory enlargement of the *pancreas* may lead to swellings in this region, but rather more often lesions of this organ producing palpable or visible swellings reveal themselves by tumours in the umbilical region, approaching the surface between the stomach, which is pushed upwards, and the transverse colon.

Aneurysm of the abdominal aorta gives a deep-seated swelling in this region, often partly under cover of the stomach or liver, usually tender, with expansile pulsation. The patient is generally a man. Diagnosis is from tumours of the stomach, pancreas, or colon, which directly transmit the aortic pulse. (See also Sect. 3, iv, p. 12.)

ii. **The right hypochondrium.** A tumour here is most often connected with the *liver*, by way either of general enlargement or of focal lesion connected with it. As this organ lies largely anterior to the lateral sagittal plane of the abdomen, its general enlargement tends to produce a visible swelling of the hypochondrium and, if extensive, of the epigastrium and umbilical regions. Liver tumours move with respiration and can often be palpated well by the bimanual method. In differential diagnosis it is important to ascertain the position and character of the lower edge, the uniformity or otherwise of the anterior surface, the degree of tenderness, and the consistency of the organ.

The *gall-bladder*, if enlarged, may present a tumour in this region—less often in the epigastric or umbilical region. The tumour usually lies in the lateral vertical plane, immediately outside the outer border of the right rectus muscle, and just inferior to the liver. Unless it be the seat of carcinoma, or contain unusually large concretions, the swelling is very elastic, rounded, elusive to the touch, generally tender, and moves with respiration. The fact that it may be of considerable size as the result of distension by mucus must be kept in mind.

The *hepatic flexure of the colon*, unless considerably dropped, can often be felt in this

region if it be the seat of growth or of scybalous masses. Such a swelling usually lies well outside the external border of the rectus, is often felt bimanually, is movable by palpation but does not move with respiration.

The tumour from which it is most difficult to distinguish a mass situated in the colon is the *right kidney*. This latter structure, however, usually gives a sense of lying more posteriorly, in addition to having features already referred to (see Sect. 3, i, p. 12). When a renal tumour is not merely the dropped normal kidney but the kidney enlarged by growth or hydronephrosis or tubercle, it is usually considerably bigger than a tumour of the hepatic colon is likely to be. It is also more definite when approached from the loin.

Suprarenal tumours yield signs very similar to, if not indistinguishable from, renal tumours. But they tend (if palpable at all) to much larger size and greater fixity, displacing adjacent organs and causing vascular disturbances.

iii. **The left hypochondrium.** By far the most common tumour in this region is the *enlarged spleen*. It lies in front of the mid-lateral plane, indeed it is usually immediately under the costal arch, emerging from beneath it with a direction downwards and inwards, its axis of elongation pointing towards the right iliac fossa. The inner (mesial) edge is more clearly felt than the outer one, and is less rounded. Along the course of this inner edge one or two notches may be felt. The tumour cannot be delimited above—an important point of distinction from a renal tumour, though not always from a suprarenal mass. A splenic tumour is much less definitely palpated by the bimanual method than is an enlarged kidney.

The enlarged *left lobe of the liver* often occupies this region, and reference has already been made to *gastric* and *pancreatic* tumours in the left hypochondrium.

Renal and suprarenal tumours give the same features on this as on the right side (see above), with an important addition—that by palpation or percussion, or both, the descending colon may often be made out lying in front of them.

The *splenic flexure*, if the seat of growth or other cause of enlargement, may be felt in this region. Owing to the fact that it is much less liable to ptosis than is the hepatic flexure, the tumour generally lies a good deal higher in the abdomen than does a tumour of the latter structure. This probably accounts for the relative infrequency with which it is palpable,

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lying, as it is apt to do, behind the stomach and spleen.

iv. **The umbilical region.**—This part of the abdomen is prone to give physical signs of many tumours which transgress their anatomical regions, particularly tumours of the stomach, liver, spleen, and gall-bladder. The tumours more proper to this region are these :

Tumours of the *pancreas* more often present themselves here than anywhere, generally as an ill-defined mass appearing between the stomach and the transverse colon. In the case of large pancreatic cysts the whole umbilical region may be occupied, and neighbouring regions to a less extent. Pancreatic tumours, whether solid or cystic, are usually very free from mobility.

Omental and *mesenteric cysts* give signs in this region more often than elsewhere.

Tumours of the *transverse colon*, unless this viscus is considerably "dropped," appear in the umbilical region, generally as rounded, fairly well-defined lumps, not moving with respiration, and usually tender.

Enlarged glands in tuberculosis and in lymphadenoma are common in this region, as also are *indurations* in tuberculous peritonitis.

v. **The right iliac region.**—Here the most common tumours are those connected with *appendix inflammation*, *new growth of the cæcum*, *the cæcal region*, or *ascending colon*, *faecal masses*, and *enlarged glands*. Differential diagnosis does not present nearly so much difficulty as in the regions of the abdomen already treated of.

Certain tumours present themselves here, however, that have to do with *organs normally lying in the pelvis*, and diagnosis in these cases is by no means always easy. These tumours include *ovarian cysts and growths*, *uterine fibroids*, diseases causing enlargement of the *Fallopian tubes* (*phlegmon*, *abscess*, *tuberculosis*), and *perimetritic and parametritic abscess*. Rectal and vaginal examinations help greatly in the elucidation of the diagnostic problems thus presented, but one of the first things to consider is whether a tumour in this region can be delimited below, or not; if it cannot, it is highly probable that, in the case of a woman, it originates in the pelvis.

Tumours belonging strictly to other regions, but which transgress so as to lie partly in this region also, are most often those of the *liver*, the *spleen*, and the *kidney*. If the kidney be very movable it may come to lie entirely here.

vi. **The left iliac fossa.**—The most common

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tumour in this region is a *cancer of the sigmoid colon*; this may lie immediately above Poupart's ligament or a little way above it.

Fæcal accumulation leads to a swelling here more often than in any other abdominal region. Enemata may be needed for diagnosis, but it must be remembered that growth plus scybulous masses constitutes a very common tumour.

Dicerculitis causes swelling in this region, rather similar in general features to a perityphlitic swelling in the corresponding right region, but less defined in outline.

All the *pelvic* tumours mentioned in connexion with the right iliac region occur on the left side also, and may be diagnosed by the same methods.

vii. **The hypogastric region.** Very few true abdominal tumours yield signs in this region. For the most part swellings felt here arise from the pelvis. Reference has already been made to the *distended bladder* and the *pregnant uterus* (Sect. 3. x, xi, p. 12).

Tumours of the *bladder and uterus*, and especially *fibroids*, occupy this region. Enlargements of the Fallopian tubes (see v and vi) may also provide swellings in the hypogastrium.

Growths of the pelvic bones, such as sarcoma, are palpable in the hypogastrium, deep seated, and confirmed often by rectal or vaginal examination.

The *urachus* lies in this region, extending from the symphysis pubis to the umbilicus. It is a common seat of secondary carcinoma, and may therefore present a definite thickening, or actual tumour, on palpation.

5. **Multiple tumours.** In the abdominal wall, multiple swellings are likely to be *lipomata*, *fibromata*, or *cancerous metastases*.

Within the abdomen, multiple swellings are most often *glandular* in nature, and perhaps occur in this order of frequency: *lymphadenoma*, *tuberculosis*, *leukæmia*, *carcinomatosis*. *Uterine fibroids* often cause multiple swellings, but these are confined to the lower abdominal zone, rarely cause ascites, and are never associated with enlargement of the liver or spleen—points of distinction from the group of multiple tumours just mentioned.

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ABORTION AND MISCARRIAGE.—The terms abortion and miscarriage are generally used synonymously, and signify expulsion of the ovum before the fœtus is of viable age. (For the legal construction of these terms,

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see ABORTION, CRIMINAL.) Medico-legally, and for the purposes of this definition, the foetus becomes viable at the twenty-eighth week of pregnancy. When the date of the last menstrual period is not available, the age of the foetus must be reckoned from its length; at the twenty-eighth week of pregnancy the foetus measures 35 cm. from the summit of the head to the heels.

Etiology.—It is approximately correct to state that about 12 per cent. of pregnancies end in abortion. The vast majority of abortions take place in the first twelve weeks of pregnancy. The etiology of abortion is imperfectly understood; it is easy to give a long list of the known causes, but it is remarkable how seldom these causes seem to apply to cases met with in actual practice. Most abortions occur in apparently healthy women, and from no ascertainable cause. The list here given—retroflexion of the uterus, fibro-myoma of the uterus, placenta prævia, accidents, acute illnesses, etc.—may be relied upon for completeness, but it must be understood that these several conditions by no means invariably cause abortion; in fact, the majority, when associated with pregnancy, only occasionally are provocative. In general terms, abortions may be placed in two groups. In the one, abnormal uterine activity is primarily at fault, from causes known or unknown; the premature uterine contractions bring about the separation and expulsion of a healthy ovum. In the other, the ovum is primarily at fault, and uterine contractions are excited secondarily by death or disease of the ovum or by intra-uterine accumulation of bloodclot.

Dealing more in detail with the causes of abortion, they may be enumerated thus:

1. *Abnormal states of the uterus*, such as retroflexion, fibro-myomata, chronic metritis, septic infections of the endometrium, double uterus and other deformities.

2. *Abnormal states of the ovum*, such as death of the foetus, vesicular mole, hæmorrhages into the chorion or decidua, placenta prævia, retro-placental and intraplacental hæmorrhages, and acute hydramnion.

3. *Abnormal states of the general health of the mother*, such as chronic renal disease (the commonest cause from the fifth month of pregnancy onwards), syphilis, cardiac disease, acute illnesses such as pneumonia, influenza, and all acute specific fevers. High temperature, cyanosis, or transmission of the disease to the foetus may be the exciting cause in

the latter group. The occurrence in cattle of epidemic abortion due to local infection with a specific bacillus (Bang) is noteworthy; abortions from a similar cause have already been reported in the human female, and further work may bring important facts to light.

4. *Traumatic causes*, such as accidents, or surgical operations on the pelvic organs or on distant parts. Women usually blame a slight accident, a railway journey, or a mental shock; but such trivial causes hardly seem blameworthy in view of the well-known fact that pregnancy often continues undisturbed after even the severest injuries and shocks.

5. The use of certain *drugs* in susceptible women, such as strong purges, quinine, anti-toxic serums.

6. *Paternal causes* are extremely rare and usually doubtful. Septic discharges in the semen may infect the endometrium; the wives of workers in lead, phosphorus, and mercury are said to be prone to abort. In syphilis the ovum is not infected directly by the semen, but always secondarily to maternal infection.

7. *Criminal abortion* is dealt with under ABORTION, CRIMINAL.

Syphilis as a cause of abortion is much over-rated. It can be demonstrated (1) that the Wassermann reaction in cases of abortion is not positive with unusual frequency, (2) that women who are known to be syphilitic do not reveal in their reproductive history a much greater number of abortions than the non-syphilitic. Syphilis is much more commonly a cause of intra-uterine death of the viable foetus (stillbirth) than of abortion. But occasionally syphilis is met with as a cause of "repeated abortion," i.e. a woman with a long series of rapidly recurring early abortions may have a positive Wassermann reaction.

Habitual or repeated abortion.—Some women abort in the early months every time they become pregnant. Usually no cause is discoverable. Among known causes of repeated abortion are retroflexion of the uterus (especially if subinvolved or fixed), syphilis, chronic renal disease, and chronic lead-poisoning (very unusual in women). Repeated abortion in the middle months of pregnancy is most commonly due to chronic renal disease.

Mechanism and anatomy of abortion. An appreciation of the outstanding features of this is of great assistance in diagnosis and treatment. The mechanism of abortion varies with the period of pregnancy. During the first two months the decidua is very thick and

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vascular, the chorio-decidual attachments are flimsy, and the ovum is small and compressible. The thick decidua has to undergo separation and expulsion as well as the ovum. In abortion during the first two months one of two courses is taken: (1) The whole ovum is expelled covered with the decidua, generally blood-infiltrated; (2) the ovum escapes first, covered with its shaggy villi, and the decidua is separated and expelled later. From the eighth to the twentieth week the placenta plays the chief rôle, and often separates with difficulty. Again, either of two courses may be followed: (1) the whole ovum is expelled intact; (2) the foetus escapes first, followed later by expulsion of the placenta and membranes. From the twentieth to the twenty-eighth week an abortion is like an ordinary labour on a small scale, and follows the same course.

Symptomatology. The first symptoms of an early abortion are painful contractions and hæmorrhage. There may be hæmorrhage but no pain, or pain but no hæmorrhage. The pain at first is slight, and may amount to little more than the sense of pelvic discomfort often felt during a menstrual period. The cervix remains closed. This early stage is called *threatened abortion*; the pain and bleeding may cease, and pregnancy may continue undisturbed. If, on the other hand, uterine contractions continue, and the cervix becomes sufficiently dilated to allow the lower pole of the ovum to be felt, or if the liquor amnii has escaped, the abortion has reached a stage when it cannot be arrested. An abortion in this stage is known as an *inevitable abortion*. It is extremely important, from the point of view of treatment, to distinguish between these two clinical stages of abortion: for in threatened abortion treatment must be directed towards arresting it, whereas in inevitable abortion treatment, if any is necessary, must be directed towards assisting it. A hard-and-fast line between the two cannot always be drawn: every abortion naturally must pass through the threatened stage, and it must be admitted that occasionally cases are met with in which an abortion has been arrested even after the cervix has become sufficiently dilated for the presenting ovum to be felt through it.

When the entire ovum has been expelled, the abortion is said to be *complete*. By an *incomplete abortion* is meant the retention of a part of the ovum, such as a portion of chorion or placenta, or even the whole placenta. When an abortion is incomplete, irregular hæmorrhage

persists, the uterus remains large and fails to involute, and the cervical canal does not close. Incomplete abortion must be recognized promptly and treated, for its course and consequences may be: (1) The retained tissue may become infected with bacteria, resulting in severe and even fatal sepsis. (2) By deposition upon a piece of retained placenta of successive layers of fibrin a fibrinous placental polypus may be formed, associated with irregular uterine hæmorrhage. (3) The retained tissue may slowly be broken up and discharged, but as a result of this process the uterus remains permanently subinvolved.

Diagnosis. When a woman is known to be pregnant the occurrence of uterine hæmorrhage and pain signifies a threat of abortion. A vaginal bimanual examination should always be made: if this is done with gentleness, there is not the remotest chance of harm being done. Other causes of hæmorrhage in early pregnancy are carcinoma, polypi, erosion of the cervix. It is stated that menstruation can occur during the first three months of pregnancy. The other outstanding condition which in early pregnancy is associated with irregular hæmorrhage and pain is *ectopic pregnancy*. In every case of hæmorrhage and pain in early pregnancy it is necessary to be on the *qui vive* for this, for a mistake in diagnosis may have very serious consequences for the patient. Cases of ectopic gestation with typical signs and symptoms are not likely to be mistaken for threatened uterine abortion; it is the cases that are anomalous in these respects and they are not uncommon which are likely to be confusing. Speaking generally, in ectopic pregnancy, recurrent attacks of excruciating pain are what the patient chiefly complains of from the start: whereas in the early stages of uterine abortion the pain amounts to little more than discomfort or, at the most, resembles miniature labour pains. But cases of ectopic pregnancy are occasionally met with in which the pain is also similarly slight. Then, again, the uterine hæmorrhage accompanying ectopic pregnancy is usually slight and thin and reddish brown, whereas that in abortion is usually profuse and contains clots. But occasionally in ectopic pregnancy the uterine hæmorrhage is more severe and is comparable to that of uterine abortion. The most reliable distinction between the two rests on the pelvic physical signs. If an enlarged tube or a pelvic hæmatocele can be felt, or if, on the other hand, nothing but an enlarged

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uterus can be felt, the diagnosis should be simple.

Cases are sometimes met, however, in which even the physical signs are misleading. To begin with, in ectopic pregnancy the uterus is enlarged to the size of a six weeks' pregnancy, and no one can be expected to distinguish between the enlarged uterus of ectopic pregnancy and an aborting uterus in very early pregnancy, in the absence of dilatation of the cervix and palpation of the ovum. Again, in cases of early tubal pregnancy the tube, although leaking from its abdominal ostium or from a rupture, may not be sufficiently enlarged to be easily palpable. Difficulty is often experienced in differentiating between a retroflexed pregnant uterus in process of abortion at about the fourth month and a large retro-uterine pelvic hæmatocele from ectopic pregnancy. In both, a large fixed swelling fills the pelvis and bulges forward the posterior vaginal wall, and in both the cervix is displaced upwards and forwards behind the top of the symphysis pubis, or even above it; in both there is, or has recently been, irregular uterine hæmorrhage and pain; in both there may be retention of urine. The following points will be helpful: (1) *The history.* - In the case of the aborting retroflexed pregnant uterus there will be a history of three or four months' amenorrhœa with no irregular hæmorrhage and no pain until near the end. With a pelvic hæmatocele the period of amenorrhœa will be much less, usually not more than six or eight weeks before the hæmorrhage and pain begin; the pain will usually have been much more severe, and the patient will almost certainly be anæmic. (2) *The signs.* - In both conditions a swelling will be found rising above the pelvic brim (the bladder, of course, being empty); in the case of the hæmatocele the upper border will be neither so evenly curved nor so well defined as in the case of the uterus, and will be resonant on percussion owing to the covering coils of small intestine. In neither case is the swelling tender. On bimanual vaginal examination the diagnosis rests essentially upon whether the uterus can be felt separately from the main swelling or whether the whole swelling is formed by the uterus. In a pelvic hæmatocele it should be possible to distinguish the uterus, pressed forwards and upwards behind and above the symphysis either in the middle line or to one side of it. The golden rule to adopt whenever the diagnosis between retroflexed preg-

nant uterus and pelvic hæmatocele is in doubt is to examine the patient under an anæsthetic. A difficult diagnosis can thus be speedily cleared up.

Treatment. This must be considered under the two clinical stages of abortion--(1) threatened, (2) inevitable.

1. **Threatened abortion.** In all cases absolute rest in bed is essential; no treatment is likely to succeed otherwise. The patient must be kept strictly in the recumbent position and must not leave her bed for any purpose whatever. As to drugs, the only ones likely to arrest the activity of the uterus are sedatives. Begin at once with a hypodermic injection of $\frac{1}{4}$ gr. of morphia, and keep the patient fairly well under the influence of the drug, given hypodermically or by the mouth, for the next three or four days. After the initial injection, the morphia may be replaced by full doses of chloral hydrate and the bromides, if preferred. The diet should be simple, and alcohol must be abstained from. If the bowels do not act for two or three days, no harm is done; naturally, strong purges and enemata are very prone to excite an irritable uterus to activity, and must not be employed. A daily simple aperient like cascara or senna must be relied upon, combined in obstinate circumstances with gentle olive oil or warm water injections into the rectum. The treatment must be continued for a week after all symptoms have gone. If the bleeding becomes severe in spite of treatment, abortion must obviously be induced.

The treatment of threatened abortion is most likely to succeed in those cases in which there is hæmorrhage only; cases in which there are also painful uterine contractions are not nearly so likely to be arrested. Even after attacks of severe bleeding in the early months, pregnancy may continue to term and result in the birth of a healthy child; and it is not at all uncommon for slight bleeding to continue, off and on, for a considerable time without the course of pregnancy being interfered with. In cases of the latter variety the question usually arises how long it is worth while allowing irregular bleeding to continue in spite of treatment (when the bleeding is too slight appreciably to affect the health). No hard-and-fast rule can be laid down, but it is approximately true that if the bleeding continues for more than six weeks there is very little chance of saving the ovum; after this period the induction of abortion may be fairly proposed or acceded to. It is important to bear in mind

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that, after the symptoms of threatened abortion have subsided, the ovum may sometimes be dead. Such retention of a dead ovum is called "missed abortion," and can only be diagnosed by noting the cessation of growth of the uterus.

2. Inevitable abortion.—It cannot be too strongly emphasized that, unless there are special indications, there is no more necessity to interfere in the course of a normal abortion than in the course of a normal labour. If the pains are strong and the bleeding is not excessive, no interference is required. A certain amount of bleeding is a necessary accompaniment of every abortion, and need cause no alarm unless it is excessive. If the bleeding is too free and the expulsion of the ovum seems rather slow, a hot vaginal douche (temperature 116° F.) and an intramuscular injection of pituitary extract or ergotin are usually effective in bringing about rapid completion of the abortion.

It is most important to examine everything that comes away, to make certain that the whole ovum has been expelled and that the abortion is complete. In this respect it is helpful to know the manner in which the ovum is accustomed to come away at the different periods of pregnancy: this has already been described under Mechanism and Anatomy (p. 15).

The indications for active interference in the course of an abortion are well defined: they are (1) excessive bleeding, (2) delay, from slow dilatation of the cervix and weak uterine contractions, (3) retention of part of the ovum (incomplete abortion), (4) infection of the uterine contents.

Active interference consists, of course, in some operation for emptying the uterus, the particular method chosen being the one most suited to the requirements of the case. The strongest emphasis must be given to three points, none of which, unless circumstances compel, must ever be omitted: an anæsthetic must be given, strict antiseptic precautions must be taken, and the intra-uterine manipulations must be gentle and deliberate, as a thin relaxed uterine wall is very easily torn or perforated.

If the course of an abortion be very slow, with the cervix scarcely dilated, or if it is decided to empty the uterus in a case of threatened abortion when the bleeding persists in spite of treatment, the best treatment is to introduce one or more laminaria

tents into the cervical canal and leave them there for twelve hours. If the cervix is then found to have dilated satisfactorily, the uterus may be emptied by the finger; or further dilatation by Hegar's dilators may be necessary before this can be done.

A word of warning may be given here about the use of the curette. The best and safest instrument to use for separation and removal of the ovum, or for a retained piece of placenta, is the gloved forefinger. By no other means can it be ascertained that the uterus is empty. It is easy, by blind groping with the ovum-forceps, to seize and tear away a piece of uterine wall, or to push the forceps through the thin relaxed uterus. Sometimes, of course, there are circumstances in which enough dilatation for the finger cannot be obtained, and in such cases the ovum-forceps must be used, but with extreme care. It is never necessary to use the curette. In emptying the uterus with the finger, it is the outside hand, over the fundus of the uterus, that has to perform most of the work, by pressing down successive parts of the uterine cavity over the tip of the inside finger, until the whole of the cavity has been dealt with. An average-sized forefinger corresponds to No. 19 on Hegar's scale.

In cases in which the bleeding has been very severe and the patient is collapsed, immediate operation is dangerous if it entails further dilatation of the cervix and separation of the ovum. In such cases it is best to plug the cervix and vagina tightly with sterile gauze, the effect of which is to excite contractions, stop the bleeding, and dilate the cervix. The plug is allowed to remain for sixteen to twenty-four hours, by which time the general condition of the patient is sure to have improved. When it is removed, the expelled ovum is usually found lying upon it, or the cervix will be found sufficiently dilated to allow the uterus to be evacuated with the finger; if further dilatation is necessary, Hegar's dilators may be used. *Plugging the cervix* is a surgical operation, and must be done with thoroughness and care; otherwise, it will not only fail to attain its object, but may cause septic infection. The bladder should be empty; an anæsthetic is not essential. Place the patient in the lithotomy position (the plugging may, of course, be done in the left lateral position, but the lithotomy is better); cleanse the vulva and surrounding skin-area with soap and water; douche the vagina with an antiseptic solution; retract the perineum and vagina with a Sims

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speculum, and pack in the gauze with a long pair of forceps (such as sponge-forceps), first filling up the cervical canal, then the vaginal vault, and then the rest of the vagina. The packing must be tight. The gauze used may be dry sterile, or soaked in an antiseptic solution. The latter is recommended, for it can be kept in the solution in a stoppered jar and conveniently fed from the jar held close to the vagina. If the pack is painful, give morphia.

The employment of a cervico-vaginal pack as a uterine stimulant and cervical dilator is not necessarily confined to cases of the above class; it may also be used as an alternative to the rapid method of dilatation by Hegar's dilators in other cases in which it is desirable to hasten the completion of an abortion. Both methods have their advantages, as well as disadvantages, and either method may be chosen for a given case, to suit the circumstances (i.e. there may or may not be at hand an assistant, an anæsthetic, the necessary instruments, etc.).

If, during the course of an abortion, it is found that the whole or part of the placenta has been retained, it is absolutely necessary to remove it at once. This is quite easily done by the fingers, with the help of an anæsthetic; no instruments are required.

In cases in which the retained placenta is infected, as indicated by an offensive odour and a raised temperature and pulse, its removal is attended by a serious risk to the patient of a general infection. Failure to remove it is, of course, attended by an even greater risk. In such cases the use of laminaria tents or a plug is strongly contraindicated. If further dilatation of the cervix is necessary, Hegar's dilators should be used, and the uterus irrigated with a copious antiseptic douche after the removal.

Preventive treatment of abortion.—The limitation of our knowledge of the causes of abortion implies a corresponding limitation of our ability to prevent it. To a woman who has aborted once, and in whom no cause can be found (*see below*), we can only give domestic or general advice. During the first four months of pregnancy she must be kept in bed during and just before and after the time of her usual menstrual periods. During the same period of time she must avoid railway journeys, motor-car rides, strong purges, strenuous exercise (riding, golf, tennis, dancing), and cold bathing or very hot baths; cohabitation must

ABORTION, CRIMINAL

be abstained from. After the fourth month is over there is small chance of her aborting in the absence of a definite cause. Cases of habitual or repeated abortion, nearly always a very unsatisfactory condition to treat, must be investigated to the best of one's ability, excluding one by one the causes enumerated on p. 15. The first thing to do is to make a pelvic examination, and exclude local conditions such as retroflexion and chronic metritis. A Wassermann reaction should be taken in both husband and wife. If a recent abortion is available, the placenta and the foetal-heart blood should be examined bacteriologically. A careful physical examination of the patient's health should be made, especially of the urine and for dental sepsis. There is little further to be done in the way of investigation. Instructions should be given that an interval of at least a year is to occur before another pregnancy, and in the meantime the health of both parents is to be brought to the highest possible level.

Of recent years success has been claimed for the treatment of abortion by preparations of the ductless glands, especially the ovary and corpus luteum. Experimental work has shown that the corpus luteum is necessary for the continued development of the early ovum. From this it is inferred that a woman may abort because her own corpus luteum tissue is insufficient, in which case it is rational to try the effect of supplying her with preparations made from the corpus luteum of cows and sheep.

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ABORTION, CRIMINAL.—The law is laid down in 24 and 25 Vic., c. 100, sects. 58, 59. Sect. 58 declares that "every woman being with child, who, with intent to procure her own miscarriage, shall unlawfully administer to herself any poison or other noxious thing, or shall unlawfully use any instrument or other means whatsoever with the like intent; and whosoever with intent to procure the miscarriage of any woman, whether she be or be not with child, shall unlawfully administer, etc., shall be guilty of a felony." Sect. 59 enacts that "whosoever shall unlawfully supply or procure any poison or other noxious thing or any instrument or any thing whatsoever, knowing that the same is intended to be unlawfully used or employed with intent to procure the miscarriage of any woman, whether she be or be not with child, shall be guilty of a misdemeanour."

ABORTION, INDUCTION OF

disease, diabetes, pernicious anemia, leukæmia, and exophthalmic goitre. (5) Diseases of the ovum such as hydatidiform mole, death of the ovum ("missed abortion"), acute hydramnios, repeated hæmorrhages in the early months, which continue in spite of treatment (see Threatened Abortion, p. 17). (6) Irreducible retroflexion of the pregnant uterus. (7) Pelvic contraction of extreme degree or obstructing fibroid tumours. Here ethical and moral standards of conduct compete. Cæsarcan section is as safe an operation as induced abortion, but patients and their relatives cannot always be convinced. There are circumstances which justify this indication. (8) Mental disease. Apart from established mental disease, cases are sometimes seen of highly "neurotic" women in whom the dread of pregnancy is so great that the question of therapeutic abortion has seriously to be considered. Such a minor mental malady, with pregnancy as the exciting cause, is extremely uncommon and is usually ended by a course of rest in bed, good nursing, massage, and sedatives. But, in spite of conscientious treatment, such patients may become so exhausted by sleeplessness, terrifying dreams, and refusal of food that their mental state becomes very precarious and renders the induction of abortion necessary.

Method of inducing abortion.—This varies with the period of pregnancy. During the first twelve weeks the uterus can be emptied, after preliminary dilatation of the cervix with tents or Hegar's dilators, with the finger and the ovum-forceps; but from this period onwards the foetus is too large to be got easily through the cervix dilated in this manner, and slower methods of evacuation, depending on the stimulation of uterine contractions, must be employed.

1. During the first twelve weeks.—One or two lamina tents are inserted through the cervix overnight. These should be about 4 in. long and must be kept in absolute alcohol for a week previous to use. They must be inserted with strict aseptic precautions. An antiseptic vaginal douche is given, a Sims speculum passed, the cervix held with a volsella, and the tent passed into the cervical canal by means of a special pair of forceps. The patient may be placed in the lithotomy or in the left lateral position; the former is more convenient for the operator, the latter more comfortable for the patient. The tent must be pushed well home so that no part projects beyond the

external os, and a small pack of gauze is used to keep it in position. It is occasionally necessary to use an anæsthetic in primigravidae with a contracted and resistant cervical canal. Next morning the patient is anæsthetized, the vulva shaved and cleansed, a vaginal douche given, the vulva, vagina, and cervix are dried and painted with iodine solution, and the tent is removed. The cervix will be found dilated and softened; the degree of dilatation, after the use of one tent, is usually such that No. 10 dilator (Hegar's scale) passes easily. Dilatation with metal dilators is now proceeded with until the forefinger can be passed into the uterus (No. 18–20 on Hegar's scale). The other hand on the abdomen presses down the uterus on to the inside finger, which gently separates the ovum everywhere from the uterine wall. When the operator is satisfied that the ovum is everywhere detached, he withdraws his finger and inserts the ovum-forceps, with which the uterus is emptied. The finger must again be passed to make sure that no part of the ovum has been left behind. An antiseptic intra-uterine douche is then given at a temperature of 118° F., and 1 c.c. of pituitrin is injected into the muscles of the outer side of the thigh. Unless the bleeding is free there is no need to pack gauze into the uterus; in fact, to pack the uterus is to invite sepsis. The vulva is dressed with the usual pad, and the patient is kept in bed for a week, assuming the sitting position from the first as much as she likes.

This little operation is simple, but requires to be done with great care.

2. From the twelfth to the sixteenth week.—

From the twelfth week onwards the head of the foetus is too large to be easily drawn through the cervix dilated in the manner described above. Induction of abortion during this period of pregnancy is the most difficult of all. It is too late to use the method described above for the first twelve weeks, and too early to use the method to be described below for induction of abortion from the sixteenth week onwards. During this middle period it is true that the cervix can be dilated with tents and metal dilators and the uterus emptied at one sitting with the finger and ovum-forceps. But such an operation is likely to tax the skill of the operator very highly. It is extremely difficult to remove the foetus, especially its head, piecemeal with ovum-forceps through a cervix which will admit not much more than the finger; such removal is made all the more

ABSCCESS, ALVEOLAR

difficult if considerable hæmorrhage is coming all the time, as is usually the case, from the placental site. A large size of curved ovum-forceps is best for the purpose, and the special forceps used for the introduction of a de Ribes bag has been found very convenient. The placenta, loosened by the finger, can usually be removed intact by gradual traction with the forceps. The finger must always be introduced afterwards, to make sure that no piece of the fœtus or placenta remains behind. A hot intra-uterine douche and an intramuscular injection of pituitrin complete the operation. The uterus should only be packed with gauze if hæmorrhage is too free and persistent.

When time permits, it is better to do the operation in two stages and to bring about a sufficient dilatation of the cervix or, better still, the expulsion of the ovum by means of the uterine contractions. The first stage consists in dilating the cervix by metal dilators, preceded or not by the insertion of a tent, sufficiently to allow the introduction of a large blunt curette, with which the ovum is broken up, making certain that the amnion is ruptured. The uterus is then lightly, and the cervix tightly, packed with gauze; at the end of about twelve hours uterine contractions will have been excited, and the operation will either be completed naturally, or there will soon be enough dilatation of the cervix to allow the uterus to be emptied with ease. The gauze should on no account be left in for more than twelve hours. If in that time pains have not begun, the gauze must be removed, a vaginal douche given, and the uterus and cervix repacked.

3. From the sixteenth week onwards.—During this period the methods used for the induction of abortion resemble those used for the induction of premature labour (*see* LABOUR, PREMATURE, INDUCTION OF). The best method is to introduce a small de Ribes bag, after a sufficient dilatation of the cervix with tents and metal dilators. The degree of dilatation necessary for the introduction of a small bag is the same as for the introduction of a forefinger (No. 18–20 on Hegar's scale).

EARDLEY HOLLAND.

ABSOESS, ALVEOLAR.—A subperiosteal abscess of the alveolar margin is a direct result of dental caries, which opens an avenue of infection from the mouth cavity to the dental pulp. The suppurative process spreads to the apex of the tooth fang and infects the socket. The cementum becomes dissolved, the tooth loosened, and pus oozes out from between tooth

and gum. Usually, at the same time, extension outwards occurs by absorption of the bone forming the outer wall of the socket, the pus reaching the external surface of the alveolar margin, where it produces a subperiosteal abscess or gumboil. Less frequently, since the inner wall of the socket offers greater resistance, perforation takes place inwards, and the abscess is found in the hard palate or inner surface of the mandible. In the case of the upper teeth the infective process may not follow either of these routes, but may spread upwards through the thin, bony floor of the antrum and give rise to an antral empyema; or it may even reach the floor of the nasal fossa from a central incisor, and cause an abscess there.

Symptomatology.—The infection of the pulp causes great pain, which continues with unremitting severity until the tension under which the pus is retained is relieved by perforation of the bone. The formation of a fluctuating swelling of the gum coincides with an amelioration of the symptoms. The affected tooth is very tender on pressure, and a carious focus will be discovered somewhere in it. The cheek is swollen and shiny. The glands on the same side of the neck are enlarged, and abscesses may develop in them. If the condition is neglected, the cheek may become adherent to the jaw and the abscess track through, eventually discharging on to the surface. In rare cases Ludwig's angina results, or thrombosis of the facial vein, with the possibility of extension from its radicals to the cranial cavity.

Treatment.—In the early stages the process may be cut short by a thorough disinfection of the tooth socket and the provision of drainage, the tooth being excavated by a dental drill—a procedure which cannot always be made tolerable by local anæsthesia. In the majority of cases the best treatment is to extract the offending tooth, removal of which is followed by a gush of pus. The abscess on the alveolus should be opened at the same time from inside the mouth; only under exceptional circumstances from the outside, even when the skin is red and the abscess near the surface. Drainage from inside may save the skin and prevent subsequent scarring. Fomentations should be applied to the face and neck. Glandular abscess may require treatment. A purge and mouth-wash (glyc. acid. carbol. 15 min. pot. chlorat. 10 gr., aq. ad 1 oz.) are always to be prescribed. Sometimes a small portion of the alveolus necroses. Should this happen the mouth must be kept clean until the

ABSCESS, AXILLARY

is loosened, which probably will not be for many weeks. No attempt should be made to remove it until this has occurred.

C. A. PANNETT.

ABSCESS, ANAL (see PERIANAL ABSCESS).

ABSCESS, AXILLARY, may be superficial when it is due to infection through the mouth of one of the sweat- or sebaceous glands of the skin of the axilla; or deep when it is due to a suppurative axillary lymphadenitis. The place of entry of the infecting organisms may be anywhere on the upper limb, chest, or scapular region of the same side. Very frequently the primary lesion is a mere scratch on one of the fingers, and has already healed by the time the patient seeks surgical aid.

Symptomatology.—First, stiffness and pain in moving the arm are felt, then a hard tender lump appears in the axilla. As the inflammation proceeds the throbbing pain becomes worse, the pus which has escaped beyond the limits of the glandular capsule being retained under tension imposed by the dense axillary fascia. There is, by this time, a large, diffuse, red, and very tender swelling in the axilla. Fluctuation may be difficult to elicit because of the tight axillary fascia. The temperature is raised. The abscess is liable to spread forwards under the pectoral muscles, backwards under the scapula, or even through the apex of the axilla into the neck. Occasionally septic thrombosis of the axillary vein develops and leads to pyæmia, while secondary hæmorrhage from the axillary artery may occur.

Treatment.—The abscess must be opened and the primary source of infection cleaned up. It is best to make the incision on the inner wall of the axilla, remembering that the long thoracic artery runs along the lower border of the pectoralis minor. A drainage-tube, not a gauze plug, should be used, and boric-acid fomentations applied. It is very important to keep the affected arm at rest in a sling, prohibiting all use of it, or healing will be long delayed. A purge must be given.

C. A. PANNETT.

ABSCESS, CEREBRAL (see CEREBRAL ABSCESS).

ABSCESS, CERVICAL.—A chronic abscess of the neck is very common, and nearly always suppurative (see ABSCESS, CHRONIC). *Acute abscess*, with which we are concerned here, is due to pyogenic infection of a lymphatic gland

ABSCESS, CERVICAL

draining some part of the head and neck or upper part of the chest. The glands most often affected are (1) the upper superficial cervical glands lying along the external jugular vein, (2) the deep cervical chain lying in juxtaposition to the internal jugular vein, (3) the superficially placed submental gland just under the point of the chin, and (4) the submaxillary gland in the substance of the submaxillary salivary gland.

Symptomatology.—Stiffness in the neck is soon followed by the appearance of a swelling. Fever occurs, perhaps preceded by a chill. At the beginning the outline of the affected gland can be defined clearly, but when the inflammatory process spreads to the periglandular tissues this definition is lost. To relieve pressure on the inflamed structure and prevent the pain of movement, the patient frequently holds his head in a position simulating torticollis. Because the abscess is so often deeply situated under the sterno-mastoid muscle and is prevented from spreading to the surface by the thick cervical fascia, fluctuation is indistinct and frequently difficult to obtain. Pain on swallowing may be complained of.

When the abscess is deep-seated in the posterior triangle the source of infection should be looked for in the mastoid region, and in the scalp as far forwards as the coronal suture. Impetigo of the scalp, septic wounds, and pediculi, even without discoverable breach of surface, are common causes.

The superficial cervical glands drain the external auricle, part of the temporal region of the scalp, part of the face, and the outer halves of the eyelids, the lymph passing, on its way, through the mastoid and superficial parotid glands. When the submental gland is inflamed a septic focus should be looked for in the middle two-fourths of the lower lip (a fissure of the lip or a pustule), or in the anterior part of the floor of the mouth under the tip of the tongue (ulcer from stomatitis, or of the frænum in pertussis). Lymph from the upper lip, inner halves of the eyelids, side of nose, outer part of lower lip, cheek, as well as a part of the tongue and floor of the mouth, flows into the submaxillary gland. The superior deep cervical glands drain lymph directly from the tonsils, pharynx, nose cavity, a great part of the tongue, the teeth, and the middle ear. Moreover, lymph from the superficial cervical glands, the submental and submaxillary glands, eventually passes into them. Hence it is that these glands are more commonly

ABSCESS, CERVICAL

affected than any others, and more especially one particular gland situated on the internal jugular vein just behind the angle of the jaw, called from its position the digastric gland or, from its frequency of involvement in disease of the tonsil, the tonsillar gland. Carious teeth, stomatitis, rhinitis, the various forms of pharyngitis and otitis, are each found as the primary lesion in deep cervical abscess. Adenoids and hypertrophied tonsils, even though they may not be inflamed, afford a free passage by means of which bacteria reach the glands in the neck.

Owing to the difficulty the pus finds in coming to the surface through the deep cervical fascia, widespread suppuration in the neck may result, and the constitutional symptoms from absorption may be very severe. Thrombophlebitis of the internal jugular vein and pyæmia and secondary hæmorrhage from the great vessels are occasional results.

Treatment.—The first thing to be done is to search for and remove the primary septic focus and so cut off the supply of invading microbes. Carious teeth must be removed forthwith, or they must be excavated, sterilized and filled. A septic focus in the scalp or face must receive attention. Enlarged tonsils, quiescent or inflamed, should not be removed until the neck condition is well, and the same qualification applies to adenoids, but a spray should be ordered for the pharynx (glyc. acid. carbol. 15 min., pot. chlorat. 15 gr., aq. ad 1 oz.). Acute or chronic otitis media must receive appropriate treatment. The *Pediculus capitis* must be eradicated. (See OTITIS and PEDICULOSIS.) Fomentations should be applied to the enlarged glands, and a massive dressing of wool, which acts as a splint, to the neck, inhibiting movements. Moist heat will not encourage suppuration if subsidence without this occurrence be possible. Should the enlarged glands increase in size, incision will be necessary, and it must not be delayed until fluctuation is indubitably present; a high temperature, an ill-defined brawniness, great tenderness, perhaps a blush on the skin, point to pus being there without the corroboration of that sign. Make the incision transversely along the direction of the natural creases of the neck, and carry it down to the deep fascia. With a dry wound and good light the deep fascia can, if it is still recognizable, be carefully divided with the knife, which is then discarded. The points of a closed Spencer Wells forceps should now be thrust into the mass and the blades

ABSCESS, CHRONIC

separated to allow of the escape of pus. If the deep fascia is softened and inflamed by the spread of the infective process, and is not recognizable, the incision should be made down to the inflamed mass and then the forceps used in the manner described. A rubber drainage-tube must be inserted and sewn in, and a dry dressing used for twenty-four hours, followed by boric-acid fomentations. The shortening of the tube must not be begun until forty-eight hours after the operation.

C. A. PANNETT.

ABSCESS, CHRONIC (*syn.* Cold Abscess).—In nearly all cases a chronic abscess is due to infection with the tubercle bacillus, though occasionally it may be due to the action of staphylococci or the typhoid bacillus. It may form wherever tuberculous granulation tissue develops, but mostly appears as the result of gland, bone- or joint-disease, a caseous mass liquefying and being converted into the so-called tuberculous pus. It is not true pus, however, but simply liquefied tissue, and contains no pus corpuscles. The softening and liquefaction are the result of the action of ferments secreted by polymorphonuclear leucocytes attracted to the necrotic tissue, and not of a secondary infection with pyogenic microorganisms which leads to acute suppuration. When liquefaction is more or less complete the fluid is of a watery consistency, of greenish-yellow colour, and contains undissolved shreds of tissue. Once formed, the abscess tends to spread by the action of gravity, guided by fascial planes. It may reach a large size, and eventually be cut off from its original source.

Symptomatology.—There are no constitutional symptoms, the patient often being unaware that an abscess is present. A swelling appears which fluctuates. The overlying skin is pale. There is no increased heat, nor is there tenderness or surrounding hardness as in a pyogenic abscess. Oedema is absent, as is also leucocytosis. If untreated, such an abscess will spread, and ultimately reach the surface, the skin at last giving way and allowing the contents to escape. Secondary infection will almost certainly take place, and the discharging abscess cavity may, as a result, never heal. On the other hand, if a case be treated properly a small abscess may be absorbed, or the contents may be removed and cure take place. Healing depends upon the resistance of the patient and on the course of the disease at the primary source.

ABSCESS, CHRONIC

Diagnosis is not always easy. A lipoma, especially in the inguinal region or back, a cyst, especially in the neck, a hernia, a hæmatoma in the scalp, may all lead to error. In the differential diagnosis the discovery of the primary bone- or joint-disease is of the greatest importance. Aspiration is decisive.

Treatment.—Complete excision is most satisfactory. This can be done in abscesses of lymphatic glands in many instances, and sometimes when the abscess is due to primary bone-disease (rib, scapula, great trochanter), the affected piece of bone being removed at the same time. Incision and drainage should never be employed; they are always followed by septic infection, and the case frequently thereby becomes incurable. Treatment for the underlying disease is essential.

Aspiration is the best treatment when total excision cannot be done. By removing the contents of an abscess, fresh serum, rich in protective substances, can be poured out into the cavity. Aspiration usually requires repetition, and may have to be performed many times before a cure is reached. A tiny skin incision is made with a knife, the skin being displaced so that this incision shall not correspond to the one in the abscess-wall. The needle of the syringe (about 2 mm. in diameter) is thrust obliquely into the abscess cavity. The site chosen should not be the most dependent part of the abscess. The needle may get blocked by caseous masses and shreds, when a stilette is useful. Sometimes the material is too thick to be aspirated. It may be made to liquefy by injecting some positively chemiotactic substance into the abscess; polymorphonuclear leucocytes then travel to the part and pour out their ferments. The best substance to inject is 2-per-cent. formalin in glycerin, prepared forty-eight hours before use (amount to be used, 5-15 c.c.). Camphorated thymol, 2-3 c.c. (camphor 2, thymol 1, ether 3), is recommended by some, but care is needed in its injection. In a few days aspiration becomes possible. The injection of 10-20 c.c. of an emulsion of iodoform in glycerin (10 per cent.) after aspiration is not employed so much as formerly.

Some surgeons prefer to lay open the abscess, wash out the cavity, wipe away caseous matter from its walls with gauze, and sew it up in layers, with or without iodoform emulsion inside. The objections to this measure are that wounds may be introduced, that the wound may not heal by first intention, and that the scar may become tuberculous. Should this last

ABSCESS, ISCHIO-RECTAL

event happen the scar must be excised and the wound sewn up again.

C. A. PANNETT.

ABSOESS, HEPATIC (see LIVER, ABSCESS OF; TROPICAL ABSCESS).

ABSCESS, ISCHIO-RECTAL.—This abscess develops deep in the fat occupying the ischio-rectal fossa. Infection with *B. coli* from the rectum is the commonest cause, the path being opened by the presence of proctitis, either gonorrhoeal or that associated with fibrous or malignant stricture, or piles. A dysenteric, tuberculous, or malignant ulcer, or a small tear at the base of a polypus, or an injury by a foreign body, may allow of the passage of the bacillus. Rarely a perforating wound from without is the cause. A tuberculous infection, especially in phthisical patients, is liable to occur via the blood-stream.

Symptomatology.—A non-tuberculous abscess is acute. It may begin quite suddenly with a chill followed by a rise of temperature, and a throbbing pain to one side of the rectum. So tender is this area, which feels hard and brawny externally, that sitting is impossible, and standing or walking gives considerable pain. These patients get most relief by lying face downwards. The movement of the bowels is very painful. Rectal examination reveals a hard, very tender mass bulging into one side of the bowel. Extension to the surface is resisted by the dense subcutaneous tissue of the buttock, but usually there is extension to the opposite ischio-rectal fossa round the posterior aspect of the rectum, a horseshoe abscess developing. Rupture takes place most often into the rectum behind, in the interval between the internal and external sphincters, and sometimes occurs both here and externally.

In tuberculous cases, unless a *B. coli* infection is superadded, a much more chronic course is taken by the abscess. In all cases alike, an examination of the chest should be instituted.

The **prognosis** is good in non-tuberculous cases, but cure may take as long as six weeks. In tuberculous or mixed infections progress is slow and recurrence to be looked for.

Treatment should be undertaken without delay, to prevent fistula formation. Under anæsthesia two incisions are made over the brawny area, one parallel to and about $\frac{1}{2}$ in. away from the anal margin, the other running out in a radial direction from the middle of this. The corners of skin should be cut away; this assists drainage. A tube is better than

ABSCESS, PERIURETHRAL

gauze. Great care in **after-treatment** is necessary. The wound should be syringed out every day with a mild antiseptic (1-in-60 phenol) and made to heal from the bottom. An enema should not be administered, purgatives being relied upon to obtain a movement of the bowels. Coincident phthisis requires attention.

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ABSCESS, LACHRYMAL (*see* DACRYOCYSTITIS).

ABSCESS, MAMMARY (*see* BREAST, AFFECTIONS OF, p. 184).

ABSCESS OF LUNG (*see* LUNG, ABSCESS OF).

ABSCESS, PERIANAL (*see* PERIANAL ABSCESS).

ABSCESS, PERITONSILLAR (*see* QUINSY).

ABSCESS, PERIURETHRAL. Etiology.

—Stricture of the bulbous urethra is the commonest cause of periurethral abscess. The mucous membrane behind the stricture is inflamed and ulcerated, and allows the passage of organisms, which produce an abscess in the perineum. Infection may also occur through a laceration made by an instrument or a breach of the surface caused by an impacted foreign body or calculus. In acute gonorrhœa one of the lacunæ of the urethra may become inflamed, with a resulting abscess. This usually occurs in the penile portion.

Symptomatology.—In the commonest or perineal form there have usually been signs of the stricture—difficulty in micturition, slow stream, and increased frequency. The development of the abscess is accompanied by pain and by the formation of a tender swelling in the perineum. Later the swelling fluctuates and the skin becomes reddened. The temperature is raised. Retention of urine from pressure is very common, and nearly always there is increased difficulty in micturition. If the condition is allowed to proceed, rupture externally and into the urethra gives rise to a urinary fistula. In the penile form acute gonorrhœa is present and the abscess develops on the under-surface of the penis.

The **diagnosis** of the perineal form is not difficult if the history be attended to. *Perianal* or *ischio-rectal* abscesses are distinguished by tracing their connexion with the rectum.

ABSCESS, POPLITEAL

Treatment should not be deferred, because of the possibility of a urinary fistula developing—a very undesirable complication. When the patient presents himself there may have been retention for some hours. No attempt at catheterization should be made, from fear of perforating the abscess-wall. Rather should all haste be made to open the abscess, when the retention will be relieved, or, if this cannot be done immediately, suprapubic aspiration should be performed. In the common *perineal* form the abscess must be opened and the treatment of the stricture postponed unless there are accompanying signs of extravasation of urine (*see* URINE, EXTRAVASATION OF). When the wound has healed, or nearly so, internal, or perhaps external, urethrotomy should be done. In the *penile* form it will generally be found that the abscess bulges into the lumen of the urethra. The best treatment is to open the abscess from the interior of the urethra through the urethroscope. Incision from the outside is said to result usually in a urinary fistula. This is by no means invariably so, and if a large penile abscess is pointing on the skin surface it should be opened from this aspect.

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ABSCESS, POPLITEAL.—Sometimes this abscess is superficial to the strong popliteal fascia and, being subcutaneous, presents no difficulties in treatment. More often it is the deep lymphatic glands lying on the popliteal vein which become inflamed and suppurate. The superficial lymphatics draining into these glands perforate the fascia with the external saphenous vein, and drain the back of the leg and outer side of the foot.

Symptomatology.—At first a stiffness in movement and then pain, which may become acute, are felt behind the knee. A hard, tender, deep swelling is palpable in the popliteal space. The knee becomes flexed, and attempts to extend it are painful and resisted by muscular spasm. Fluctuation is difficult to perceive because of the dense popliteal fascia over the abscess, and at first the overlying skin is not reddened. The pus being under tension, constitutional symptoms may be pronounced. The infective process, extending through the gland capsule, may give rise to a large abscess in the popliteal space which, pressing upon the popliteal vein, causes œdema of the whole leg and foot. Rarely, septic thrombosis of the vein takes place, leading to pyæmia. In the abscess breaks through the popliteal space and

ABSCESS, PREPATELLAR

becomes superficial there is a distinct amelioration of the symptoms.

Treatment.—Early incision is necessary, and the same care should be taken as in opening cervical abscesses. Over the most prominent part of the swelling a vertical incision is made. It must, however, be to one side of the middle of the popliteal space to avoid injury to the external saphenous vein. It reaches in depth the popliteal fascia, which should then be divided cautiously with the knife. The points of a pair of Spencer Wells forceps are now pushed through the capsule of the gland and opened, allowing the pus to escape. The popliteal fascia may be infiltrated and indistinguishable. If so, resort to the forceps must be made earlier, for it will be easy to penetrate the softened fascia. A tube is to be sewn in and left for forty-eight hours before shortening is begun. The patient must be in bed. Great care must be taken that the tube does not press unduly on the popliteal vessels, for secondary hæmorrhage, resulting in death, has thus been caused. For this reason the tube must not be left untouched for longer than forty-eight hours. The source of infection should receive treatment.

Another, and in many respects a preferable method of reaching the pus, inasmuch as it is fraught with less risk of injury to important structures, is to approach the abscess from the inner side. An incision is made parallel to and just behind the adductor magnus tendon, beginning about $\frac{1}{2}$ in. above the adductor tubercle, and reaching upwards for $3\frac{1}{2}$ in. The anterior border of the sartorius is defined and this muscle retracted backwards. The adductor magnus tendon is then pulled forwards, and behind this the semimembranosus is dragged backwards. The abscess is opened by thrusting the points of a closed Spencer Wells forceps into the floor of the wound. Access is thus gained to the popliteal space by the same route as is followed in tying the popliteal artery, and without encountering any important nerves or vessels. C. A. PANNETT.

ABSCESS, PREPATELLAR.—An abscess developing in the prepatellar bursa—a suppurative bursitis.

Etiology.—Infection may take place by a direct wound of the bursa, by spread from a small abscess, pustule, or abrasion superficial to it, or as a result of an infective process in the leg or foot. In connexion with the last method it is to be remembered that the bursæ

ABSCESS, RETROPHARYNGEAL

constitute part of the lymphatic system, and that the lymphatic vessels open into them. Thus, organisms passing up the lymphatics from a nidus in the foot or lower leg can cause an inflammation of the prepatellar bursa, especially if there has been constant irritation of this structure from kneeling.

Symptomatology.—Pain is felt in front of the knee, and a tightness, which limits flexion. The bursa becomes distended with fluid. The upper limit of the swelling corresponds with the middle of the patella. The exudation, which is serous at first, soon becomes turbid and then purulent. The overlying tissue is red and œdematous. Pus, breaking through the confines of the bursa, leads to a peripheral cellulitis. Owing to the resistance of the thick periosteum in front of the patella and the stout capsule of the knee-joint, spread of the disease to bone or joint is very unusual. The overlying skin eventually gives way, and the pus is discharged externally.

Diagnosis is easy, but care must be taken not to mistake a serous effusion into the bursa, due to a neighbouring septic focus outside the bursa, for a suppurative bursitis. If such a bursa be opened the wound may fail to heal, at least for a very long time, and continue to exude a clear serous fluid (bursal fistula). In cases of doubt an aspirating needle should be employed.

Treatment consists in opening the bursa. Two vertical incisions should be made, one on each side, so that when healing occurs there is no pressure on the scar in kneeling. A drainage-tube is inserted and boric-acid fomentations applied. It is best to keep the patient in bed, when a back-splint will be unnecessary. The originating focus must receive attention. Should a fistula persist, owing to the endothelium around the margin of the incision escaping destruction, it may be made to close by opening the bursa widely and stuffing it with gauze until its inner surface is lined with granulation tissue, or by destroying its endothelial lining with pure liquid carbolic acid.

C. A. PANNETT.

ABSCESS, PSOAS (*see* SPINAL CARIES).

ABSCESS, RETROPHARYNGEAL, may be acute or chronic in type. *Acute* suppuration is due to pyogenic infection of the retropharyngeal lymphatic glands secondary to adenoids, diphtheria, scarlet fever, or other naso-pharyngeal infection. The abscess lies in

ABSCESS, RETROPHARYNGEAL

front of the prevertebral fascia. Suppuration in the parotid, or even in the middle ear, will sometimes extend inwards to the postpharyngeal space, causing an acute abscess in this situation. Such abscesses are most common in children. The onset is sudden, with pain in the back of the throat, rigor, and high temperature. The head is fixed and thrown somewhat back, with the mouth open. The tongue is slightly protruded to lessen the difficulty in breathing. Respiration is accompanied by a crowing sound. Swallowing is impossible, and saliva drains out of the mouth. The crying is muffled. On inspection a swelling is seen on the postpharyngeal wall to one side of the middle line; the palate is pushed forward. The sensation of a tense bag of fluid is obtained on palpation with the finger-tip, a method of examination without which the diagnosis is often missed. If left alone the abscess ruptures into the pharynx. Death frequently results from suffocation, or from the subsequent development of septic pneumonia. The *chronic* form is secondary to cervical caries or a tuberculous infection of the glands. The spinal abscess lies behind the prevertebral fascia. There are not the constitutional signs or the distress of the acute cases. The head is thrown back with the chin protruded, or it is in the position of torticollis. The neck is rigid. There may have been the radiating root-pains of spinal caries. The child often sits supporting its chin on its hands. If the abscess is due to tuberculosis of the retropharyngeal gland there are always other tuberculous glands in the neck.

Treatment.—In *acute* cases the abscess should be opened from the mouth. Except in very extreme cases, chloroform may be administered, but anaesthesia should be light. The child is held on one side with the head lowered and the feet raised. A scalpel guarded with strapping to $\frac{3}{4}$ in. from its point is used, or sinus forceps may be thrust into the abscess. With ordinary care pus will not get into the larynx. In *chronic* cases the opening should never be made from the mouth. The abscess may be reached from the neck behind the sternomastoid and the carotid sheath. The line of incision corresponds with the posterior border of the sternomastoid; it should be above the middle of this border in order to avoid injuring the emerging spinal-accessory nerve. Its cavity is opened, washed out, and the wound sutured completely. But if an examination of the pus shows that septic organisms are present a drainage-tube must be left in. Treatment for

ACANTHOSIS NIGRICAANS

the spinal disease should be instituted at once. Tuberculous glands in the neck can be removed.

C. A. PANNETT.

ABSCESS, SUBMAXILLARY (*see* ABSCESS, CERVICAL).

ABSCESS, SUBPHRENIC (*see* SUBPHRENIC ABSCESS).

ABSCESS, TROPICAL (*see* TROPICAL ABSCESS).

ACANTHOSIS NIGRICAANS.—A very rare disease characterized by irregular pigmentation and a rough and warty condition of the skin. the latter chiefly in the folds and flexures.

Etiology and pathology.—The condition was originally described by Pollitzer and Janowski in 1890. In this country its recognition has been mainly due to Sir Malcolm Morris. Women appear to be more often affected than men, and while the disease may occur at any age, it is observed more often in the third and fourth decades. The coexistence of cancer in the abdomen is a common but not invariable feature of the adult cases. Histologically, hypertrophy of the epidermis is observed, and an excess of pigment, especially in the lowest layer of the rete Malpighii, together with collections of mast cells; but such appearances are characteristic rather of the various conditions which make up the disease than of the disease itself. Pigmentation being so striking a feature of the disease, attention has naturally been directed to the suprarenal glands and semilunar ganglia, but, although pathological conditions of these structures have occasionally been found, they are quite inconstant.

Symptomatology.—More or less deep pigmentation, a furrowed or morocco-leather condition of the skin, and warty formations, mostly blackish, are constant features; the lesions are symmetrical and regional in their distribution. The pigmentation is not universal, occurring, according to the observations of Morris, chiefly on flat surfaces and certain flexural regions. It is important to note that the mucous membranes escape pigmentation. In consequence of an exaggeration of the natural lines of the skin a condition of roughness is produced, and here again an important negative fact—the absence of scaliness—is to be observed. The warts or papillomata, which may be either sessile or pedunculated, are not necessarily associated with the pigmented areas, but are found

ACHONDROPLASIA

affecting certain localities with a considerable degree of regularity. According to Darier, the ano-genital region and the nape of the neck are first involved; subsequently, the axillæ, the umbilicus, the hands, the bends of the elbows, the regions about the eyes and mouth, the feet and popliteal hollows. In this process the mucous membranes frequently share, and the tongue exhibits a curious villous appearance. Nails and hair are usually abnormal.

Diagnosis is easy in a typical case. Addison's disease, Darier's disease, and arsenical pigmentation should be excluded.

Treatment is unsatisfactory. All that can be done is to remove the warts surgically, and to give emollients and alkaline baths to help the general condition.

H. MACCORMAC.

ACCOMMODATION, ERRORS OF (*see* REFRACTION AND ACCOMMODATION, ERRORS OF).

ACETONURIA (*see* URINE. EXAMINATION OF).

ACHOLIA (*see* MORBUS CÆLIACUS).

ACHOLURIC JAUNDICE (*see* JAUNDICE, Vol. II, p. 146).

ACHONDROPLASIA (*syn.* Chondrodys-trophy; formerly, and erroneously, Fœtal Rickets).—In achondroplasia, from some unknown cause acting from the third to the sixth month of fœtal life, ossification in the primary cartilage of bone ceases, with the result that there is interference with the development of the long bones of the limbs, the ossa innominata, ribs, and basi-occipital. The condition is about equally common in males and females. It is rarely hereditary or familial. Achondroplasias seldom have children.

Symptomatology.—Achondroplasia is a congenital, lifelong, and irremediable condition, producing in typical cases a most characteristic picture, in which the limbs are dwarfed out of proportion to the rest of the body (*cf.* dachshund dog). In stature the full-grown achondroplastic is not more than 4 ft. high, owing chiefly to the dwarfing of the lower extremities. The trunk would, however, be normal for a person of greater height save that, owing to some tilting of the pelvis, there is usually considerable lordosis. The head is large in proportion to the height, and somewhat hydrocephalic in shape. In face, all achondroplasias are very much alike: the forehead large and prominent, the nose depressed,

ACNE ROSACEA

the nostrils rather wide, and the jaw a little overhung. The teeth and the mouth are usually normal. The limbs are disproportionately short. This is particularly noticeable in the upper arms and thighs, where the bones are also considerably curved. The gait is waddling in character, and the knees have the appearance of being dislocated. When the patient is standing upright with the arms to the sides, the finger-tips do not reach lower than the great trochanters. The hands are small. The fingers are unduly equal in length, and when extended their tips are separated (ray-like hand). The middle and ring fingers are particularly separated, giving the characteristic *main-en-trident*. Muscular development is usually good. The sexual organs as a rule develop normally. Mentally achondroplasias are not of a high level of intellect, but are usually sharp and shrewd.

Diagnosis.—In a typical case the diagnosis is easy. It rests chiefly upon the disproportionate stunting of the limbs. The condition may be mistaken for hydrocephalus, rickets, and dwarfism, but in none of these are the limbs so short in proportion to the height of the patient. On the other hand, there may be ill-marked cases, associated perhaps with additional rickety changes, in which the diagnosis may be difficult.

Prognosis.—Many achondroplasias die soon after birth or during early infancy. Should this period be successfully passed the condition does not tend to shorten life. In the event of an achondroplastic woman becoming pregnant, (æsa-rean section gives the only chance of obtaining a living child. No treatment modifies achondroplasia.

REGINALD MILLER.

ACHROMATOPSIA (*see* COLOUR VISION).

ACHYLIA GASTRICA (*see* STOMACH, FUNCTIONAL DISORDERS OF).

ACID DYSPEPSIA (*see* STOMACH, FUNCTIONAL DISORDERS OF).

ACIDOSIS (*see* DIABETES MELLITUS).

ACNE ROSACEA.—Rosacea, as this condition is more usually called, is the name given to a chronic reflex congestion of the central part of the face, leading to permanent vascular dilatation, follicular inflammation, and hypertrophy of the sebaceous glands and connective tissues.

Etiology and pathology.—The disease is essentially one of middle life, though cases

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occur in younger people. It is also much commoner in women than in men. It is primarily a vascular congestion of the face. The congestion is in all cases a reflex one, and in the vast majority of cases is the result of chronic dyspepsia. It is common in women about the menopause, and also in alcoholics, but in both these cases the primary cause may well be an associated dyspepsia. The congestion is followed by a mechanical dilatation of the superficial vessels and also, owing to the increased blood supply to the part, to hypertrophy of the sebaceous glands, followed in some cases by hypertrophy of the connective tissue of the skin and subcutaneous tissues. As a result of the sebaceous-gland hypertrophy, secondary staphylococcic infection is liable to occur, with the production of follicular pustules which resemble *acne vulgaris*, though there is no comedo formation. It is not clear whether the hypertrophy of the connective tissues is produced by the vascular changes alone, or whether other factors, such as lymphatic obstruction, also play a part.

Symptomatology.—The earliest symptom is an intermittent flushing of the face, which subsequently becomes persistent. The area involved includes the forehead, chin, nose, and adjacent parts of the cheeks. After the flushing has persisted for a time, definite dilated vessels can be seen, and these are especially apparent on the nose near its tip and on the alæ. In some cases these changes are associated with great patulousness of the sebaceous follicles, and an excessive oily secretion, rendering the face shiny and even obviously greasy, but in others this excessive sebaceous secretion is absent. There is a pronounced tendency to the formation of perifollicular red papules, and these not infrequently show a central pustule. Occasionally deep-seated nodules similar to those found in *acne vulgaris* are present, but are less frequent than in that condition. Conjunctival injection and even corneal ulceration are occasionally concomitant symptoms.

In cases in which there is not much seborrhœa the follicular lesions are not usually present, but a dry scaly dermatitis may develop, especially in those much exposed to the sun and wind.

In some cases the redness is confined to the nose, and it is in these that hypertrophy of the connective tissues is particularly liable to occur. In the earlier stages of this condition the tip of the nose becomes wider

than normal, but, later, tumour-like masses with greatly dilated sebaceous follicles appear on the tip and alæ of the nose, producing the condition known as *rhinophyma*. This condition occurs much more frequently in men than in women, and usually in alcoholics and those exposed to the weather, but this is not always the case. A rare type has been described in which the hypertrophy occurs in the skin over the glabella, and it is probable that some of the cases of chronic solid œdema of the face, especially in that position, are of a rosaceous nature.

Diagnosis.—The hypertrophic type is so characteristic that it cannot be taken for anything else. The acneiform type, especially when it occurs in young people, may be mistaken for *acne vulgaris*, but the presence of the underlying congestion and the absence of the comedo in rosacea should suffice for a correct diagnosis. It must be remembered, however, that the two conditions may occur simultaneously. In the drier forms, in which the whole area described above is involved in the congestion, a diagnosis from *lupus erythematosus* may have to be made. In the latter condition the edge is well defined and raised, the patch is infiltrated, and the scales are very adherent, while in the centre atrophic scarring may be seen; but in rosacea none of these things is present.

Prognosis.—Early cases under suitable treatment recover quickly, but once the disease is well established and much hypertrophy or many telangiectases are present, a complete cure is difficult to effect.

Treatment.—The main treatment of rosacea must be directed to the underlying cause, which, as noted above, is chiefly dyspepsia. This is generally of the atonic, flatulent type and is associated with hyperacidity. Meals should be taken with as little fluid as possible, and the quantity of vegetables, and especially of green vegetables, reduced to a minimum; alcohol and strong tea and coffee should be interdicted, as well as hot and highly seasoned dishes. A tumblerful of hot water taken while dressing in the morning, and a similar quantity an hour before the mid-day and evening meals, should help to avoid the necessity of large potations with the meals. If constipation is present, an effervescing saline aperient may with advantage be added to the morning glass. The importance of fresh air is obvious. Great benefit is obtained by the administration of sodium bicarbonate (15–20 gr.) three times

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a day before food in a simple bitter; while in some cases the exhibition of intestinal antiseptics, such as ichthyol (5 min.) or creosote (1 min.) in capsules three times a day, is found to be of considerable value. Locally, in the congestive stage, a simple sedative lotion is all that is required; lotio calaminæ answers the purpose well, and in the cases associated with much seborrhœa the addition of 5 per cent. precipitated sulphur is useful. In these cases, too, the face should be washed two or three times a day with borax and camphor soap and tepid water. In the dry scaly cases soap and water should be used sparingly, not more than once a day, and a basic superfatted soap used, followed by the application, after careful drying, of a simple cream such as ung. aquæ rosæ. It is very inadvisable in the congestive stage to use strong applications, as they tend to increase the congestion. The pustules rarely require special treatment, but if deep-seated they may be opened and evacuated. In the telangiectatic stage it is advisable to destroy the superficial vessels, either by a fine-pointed cautery or by the electrolytic needle; the former is much more rapid and, if a sufficiently fine point is used, is less painful than the latter. When large connective-tissue overgrowths occur, the redundant tissue may be removed freely with a knife.

A. M. H. GRAY.

ACNE SCROFULOSORUM or Cachectiorum (see TUBERCULIDES).

ACNE VULGARIS.—A follicular and perifollicular inflammation of the sebaceous glands, and their ducts, usually limited to those of the face, shoulders, chest, and back.

Etiology and pathology.—Acne vulgaris is essentially a disease of the adolescent, beginning most frequently about the age of puberty, and is not common after the age of 30. It affects both sexes about equally, and is as frequent among the well-to-do as among the poor. It almost invariably occurs in those who suffer from oily seborrhœa, which may be looked upon as a predisposing cause. The earliest stage is the formation of the comedo or blackhead; a putty-like plug filling and distending the pilo-sebaceous follicle, and is visible to the naked eye as a black spot at the mouth of the follicle. This plug can easily be expressed, when it is found that only the portion filling the mouth of the follicle is of black colour, due to dirt and

degeneration of the horny substance forming the plug, and that the main mass is cream-coloured. On microscopical examination the plug will be found to consist of epithelial cells and sebaceous material, together with numerous micro-organisms; in some cases a small acarus, the *Demodex folliculorum*, which appears to have no etiological significance, is present. The organisms usually found are three in number—a small bacillus, the *Bacillus acnes*, which permeates the whole comedo; a yeast-like organism, the so-called bottle bacillus, and the white skin-staphylococcus, both of which are chiefly found at the mouth of the follicle.

The comedo is the result of a chronic inflammatory process occurring in the follicle, resulting in an excessive formation of horny cells; these, mixed with sebaceous secretion, which cannot escape freely, form the comedo. It is generally held that this inflammatory process is due to the *Bacillus acnes*, but this is not yet completely established.

Subsequently to comedo-formation a perifolliculitis or periaidentitis may be set up, with or without pus-formation. It is probably due in the majority of cases to secondary infection by the staphylococcus, though in some cases the *Bacillus acnes* appears to be responsible. Whichever organism is concerned, however, there is another factor which plays an important part, namely, the resistance of the patient; in other words, some subjects are much more prone to suppurative complications than others.

Symptomatology.—The majority of adolescents show some signs or other of acne vulgaris. In the mildest cases only a few comedones may be seen, whilst in the extreme forms most disfiguring inflammatory lesions are present. The lesions are usually limited to the face, shoulders, chest, and back, but in the extreme cases may involve the ear, side of the neck, and upper part of the arms. The centre of the face is most frequently affected, and it will be noted that the lesions are not confined to the central area as in acne rosacea, but affect the whole face. In the mildest cases just a few comedones may be seen, and these chiefly at the angles of the nose, on the cheeks, and on the forehead; the forehead is particularly implicated in girls who allow their greasy hair to lie over the forehead, and in boys who wear greasy caps. In one type of case these comedones may be found scattered all over the face in large numbers, almost



Fig. 1.—Punctate, papular, and pustular types of lesions.



Fig. 2. Nodular lesions and pitted scarring.

PLATE 1.—ACNE VULGARIS.

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every follicle being involved, but without showing any inflammatory signs; this type is called *acne punctata*. In the most common type the eruption on the face has a polymorphic character (PLATE 1, Fig. 1); the lesions vary from a dozen or two up to several hundred; some of them are the ordinary comedo, and others show a central blackhead with a small reddish zone around; in yet others there is a large, lentil-sized red prominence with a central pustule. Acne of this type has been named *acne papulosa* or *acne pustulosa*, according to the type of lesion which predominates. In another group of cases deep-seated nodules are present, associated with the lesions just mentioned; they appear as firm lentil- or pea-sized nodules, situated deeply in the skin, and not always obviously connected with a comedo (PLATE 1, Fig. 2). As they enlarge, the skin over them becomes first a purplish and then a dull-red colour; later, the surface skin becomes thinned and definite fluctuation may be felt; and finally, a point of pus may be seen in the centre of the inflamed area. If they are opened or allowed to burst, either definite pus or a glairy yellow fluid may escape. These lesions vary much in character; sometimes they run an acute course like a superficial abscess, developing rapidly with a good deal of local tenderness, discharging pus, healing, and leaving a well-marked scar; in other cases the deep-seated nodules never develop but gradually subside without suppuration, whilst still others may slowly form large painless fluctuating swellings with characters resembling sebaceous cysts. The lesions may occur in large numbers, and generally produce considerable disfigurement, both during the active stage of the disease and also subsequently from the scarring they leave; this is particularly the case if the scarring becomes cheloidal, as sometimes happens. This type of case is spoken of as *acne indurata*, and is generally much more persistent than the papular and the pustular types; the eruption is frequently most pronounced on the back, which may be involved in its entire length down to the upper part of the buttocks, and the lesions often remain to a much later age than in the other types.

The course of the disease may extend over several years. In the milder cases there are usually periods of comparative freedom from lesions, recurrences being often dependent on variations of the patient's health, especially in the nodular type; while in the more

severe cases the patient is rarely free from lesions as long as the disease lasts.

Diagnosis.—This presents few difficulties if the points mentioned above are kept in view, namely, the age of the patient, the limitation of the acne lesions to the face, shoulders, chest, and back, the multiformity of the individual lesions, their acute course and rapid cure when the pus from the follicle is evacuated, their tenderness when the inflammation is at its height, and finally the presence of the comedo in nearly all the lesions. The condition will rarely be confused with *acne rosacea*, in which the hyperæmia of the face is the prominent feature, the lesions are almost entirely limited to the central portion of the face, and the comedo is not found in the follicles; in addition, the pustules are usually painless and contain very little pus. Rosacea, again, is much more common in middle life than in adolescence, though it does sometimes occur in persons between 20 and 30, who may have a pre-existing acne vulgaris; in these cases, however, there should be no difficulty in distinguishing the characteristic lesions of the two diseases. The acne sometimes produced by the internal administration of *iodides* and *bromides* is easily distinguishable, as a rule, from acne vulgaris; the pustular lesions are much less symmetrical, and tend to be grouped into tumour-like masses; the face is frequently affected, but the chest and back rarely, while lesions not uncommonly appear on the extremities, sites never involved in acne vulgaris. Bromides, however, appear in some cases to exaggerate a pre-existing acne vulgaris. Tertiary *nodular syphilides* sometimes have an acneiform distribution; they are not, however, essentially follicular and do not become pustular; they are infiltrated, painless, brownish-red nodules, and are very persistent if not treated. The presence of a positive Wassermann reaction and the rapid disappearance of the nodules under iodides or salvarsan will confirm the diagnosis.

Prognosis.—The condition disappears spontaneously in the course of years, but, especially in the nodular cases, may leave very disfiguring scars if not promptly treated.

Treatment.—Acne vulgaris and the underlying seborrhœa are so frequently associated with derangements of the general health that appropriate treatment of these conditions is necessary. Dyspepsia, constipation, and anaemia are most commonly met with. The dyspepsia of adolescents is chiefly due to immoderate

ACNE VULGARIS

eating, the improper mastication and bolting of food, and an excessive sugar and carbohydrate diet. Constipation and anæmia may also result from this, but are also met with in those whose occupations are sedentary and who work in crowded and badly ventilated rooms. In girls menstrual disturbances may play a part. To deal with these conditions, regular meals should be insisted upon, a simple mixed diet given, and plenty of time allowed for proper mastication. It should be unnecessary to give drugs for this dyspepsia: a properly regulated diet should suffice in an ordinarily healthy young person. If, however, the constipation is not corrected by diet, liquid paraffin or saline aperients are indicated. If anæmia is present, iron or arsenic should be given. Many of the menstrual disturbances of young girls are secondary to anæmia, and should be dealt with by the exhibition of the same drugs. Exercise and plenty of fresh air are useful adjuncts to prevention and treatment.

Local treatment is, however, called for. A great deal can be done to prevent the occurrence of the comedo: persons with abnormally greasy skins should take frequent hot baths, and, after drying with a rough towel so as to empty the follicles of sebaceous matter, should dust on a little talc powder containing 2 per cent. of precipitated sulphur. The same procedure may be adopted after the comedones have formed, but in this case it will be necessary to squeeze out the comedo with an extractor or, better still, by the fingers covered with a handkerchief. Ointments should generally be avoided, but may be necessary in the more severe cases. If suppuration has occurred, the pustules should be opened under aseptic precautions and the contents evacuated. In the nodular variety with skin abscess, small incisions with a fine scalpel should be made, the contents expressed, and the cavity painted with pure carbolic acid.

Vaccines have been largely employed of late years in treating pustular acne, but with very varying results. Vaccines of the acne bacillus and the staphylococcus, or mixtures of the two, have been used. The best results are obtained in cases in which the staphylococcus is responsible for the pus-formation. The most satisfactory treatment, however, is radiotherapy. In chronic nodular cases the results are extremely satisfactory. On the trunk one usually gives a single full Sabouraud-pastille dose to each affected area; to the face, either a single

ACTINOMYCOSIS

dose of $\frac{1}{2}$ pastille, or $\frac{1}{3}$ pastille weekly for three or four weeks, a screen of 0.5 mm. aluminium being employed in either case.

A. M. H. GRAY.

ACROMEGALY (see PITUITARY GLAND, AFFECTIONS OF).

ACROPARÆSTHESIA.—Subjective sensations of numbness and tingling, or "pins and needles," in the extremities, particularly in the hands and fingers, is a not uncommon affection in women in middle life. The term acroparæsthesia should not be applied to those cases in which demonstrable disease is present, which may produce sensory phenomena as an initial symptom. Such conditions are tabes and other spinal diseases, cervical rib, chronic neuritis, as in diabetics, and gross vaso-motor diseases such as Raynaud's disease, endarteritis obliterans and arterio-sclerosis. Excluding such definitely organic diseases, the symptoms of acroparæsthesia are apparently due to a vaso-motor neurosis, though such vaso-motor phenomena as cyanosis, pallor, mottling, and œdema are usually conspicuous by their absence. Indeed, the symptoms are in a large majority of cases entirely subjective. Many causes have been ascribed for their development, particularly the climacteric. Chronic gastritis and anæmia are other common causes; yet others are chronic rheumatism, especially affecting the fingers, and exposure to cold from having the hands frequently in water, as in washerwomen. The sensations are almost entirely limited to the fingers, and hardly ever involve the feet, and the paræsthesiæ are usually continuous, though worse at night and increased by use of the hands. Definite anæsthesia is never present, though there may be some slight apparent blunting of sensibility. The disease, if such it may be called, runs a chronic course, and may last for years, though the symptoms may sometimes be improved by local massage, faradic or galvanic baths, and tonic treatment with arsenic, iron, and phosphorus.

WILFRED HARRIS.

ACTINOMYCOSIS.—A chronic or sub-acute inflammation, chiefly affecting connective tissues, induced by infection with the actinomyces (PLATE 28, Fig. 8, Vol. III, facing p. 138), and leading to a formation of pus containing characteristic minute granules of the fungus.

Etiology.—There are many forms of streptothrix infection which are pathological to

ACTINOMYCOSIS

animals or man, but there is reason to believe that most of the cases of human infection are due to one particular type of organism. This streptothrix is a Gram-positive organism which grows well on glucose-agar under aerobic or, preferably, anaerobic conditions. There is often a small coccus also present in the actinomycotic lesions. The causative organism is parasitic on various grasses and cereals, hence it is easily understood that herbivorous animals are prone to the disease; it is especially common in the tongue and submaxillary region of cattle.

Actinomycosis is by no means a rare condition, though there is a prevailing impression to the contrary. It is liable to be overlooked and wrongly diagnosed unless the observer makes a point of always considering streptothrix infection as a possibility in every case of chronic inflammation.

The condition is not found in very young children; the majority of the cases occur during the second, third, and fourth decades of life. More males than females are attacked. Though from the habitat of the fungus one might conclude that those living in country districts would be more subject to the disease than the inhabitants of towns, yet the condition appears to be as common among the urban population. Debilitating conditions are stated to predispose to the infection, but it must be allowed that most of the cases occur in people who are otherwise quite healthy.

The *mode of infection* is in many cases uncertain. Direct infection from animals is probably rare. The fungus, which normally lives on vegetable matter, is most probably conveyed to human beings by this means. Many cases are recorded in which a grain of barley or a fragment of grass has been found at the centre of the lesion. In most cases, one must assume, a minute portion of the fungus insinuates itself into the tissues through a surface abrasion. Such abrasions are readily caused in the mouth, which is a common path of infection. In thoracic actinomycosis the organism is probably conveyed to the part by inhalation of minute particles of the fungus contained in dust.

Pathology.—The pathological changes induced in the tissues by the fungus are those of subacute or chronic inflammation of an indurative type. Soon after infection an inflammatory reaction leads to the formation of soft connective tissue, which often becomes more dense until it is converted into fibrous tissue as

hard as wood. Occasionally the characteristic hard tissue is not formed, but the pathological condition is represented by a mass of soft vascular granulation tissue containing a large amount of the fungus, which appears as yellowish streaks or specks. The only bone commonly affected is the mandible, which may be much enlarged and riddled with tracks lined with granulation tissue.

Rarely, extension of the disease by way of the blood-stream may occur, abscesses being formed in organs such as the brain or kidney without any sign of induration.

Symptomatology and clinical types.—

The lesions of actinomycosis usually evolve somewhat more slowly than those of tertiary syphilis, but more quickly than those of tubercle. The pain experienced is often slight, and the indurated form is painless. The hard, wooden mass of inflammatory tissue which so frequently develops is characteristic, and, when considered with the absence of pain and tenderness, presents a picture which is like nothing else in the whole range of surgery.

The inflammatory process prefers to attack connective tissues, seldom invades the serous sacs, and avoids the lymphatic glands in a truly remarkable way. Sooner or later, softening always occurs, and the typical granules of the fungus appear in the pus which is evacuated. Secondary infection is common.

In the human body there are three sites of election for the development of the streptothrix of actinomycosis. First and foremost is the region of the mouth and face, with the cellular-tissue planes around and communicating with this area. The thorax and the caecal region of the abdomen are the other two common sites. In addition, the disease is likely to develop at any superficial site of inoculation in the skin or subcutaneous tissues.

In four cases out of five infection occurs through an abrasion of the lining of the *buccal cavity*, either tongue, gums, or tonsils. Occasionally infection occurs along the salivary ducts, and possibly carious teeth allow access to the interior of the mandible. Soon a rounded swelling is formed in the parotid or submaxillary region. If the process is rather acute the swelling may be painful, but in most cases pain is only slight. If the face and parotid regions are affected the swelling shrinks and becomes firmer and tends to migrate very slowly to the region of the angle of the jaw. In two or three months the hard mass begins to soften, and a sinus forms just behind the angle of the jaw.

ACNE VULGARIS

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From the discharge may be obtained the typical granules of fungus. The infiltrated area tends week by week to spread down the neck, forming new sinuses, while the original ones close up. Thus arise the puckered cicatrices so commonly associated with the condition. In some instances there is formed a big mass of tissue as hard as wood, which may persist for years but only discharge slightly; in other cases softening, sinus-formation, and puckering are more in evidence.

In the *submaxillary region* the disease causes a similar diffuse, hard, chronic painless swelling. Infection is sometimes seen lower down the neck, a position probably reached by continuity of process, the original focus having healed. Sometimes a nodule occurs in the *tongue*. The lymphatic glands in the neck do not become enlarged and apparently are not affected except in the general mass. The horizontal ramus and angle of the *lower jaw* may be involved and become greatly enlarged by the formation of much soft cancellous tissue riddled by sinuses.

When actinomycosis attacks the *cæcal region* the clinical symptoms may be ushered in acutely or have an insidious onset. When acute the initial symptoms are those of an ordinary acute appendicitis with perforation and local abscess-formation; after the abscess has been drained, healing does not take place so quickly as is normal, and the sinus track becomes indurated. This induration extends until a hard lump can be felt in the iliac fossa. Ultimately softening occurs in some part of the mass.

Cases with gradual onset first come under observation with a hard lump palpable in the right iliac fossa. Advice may be sought for some symptom which at first seems unconnected with the abdomen, e.g. pain down the thigh. If the *psoas* is infiltrated the hip may be kept flexed. Abdominal pain is not usual. It is very rarely that the peritoneal cavity is invaded. The process extends retroperitoneally upwards towards the diaphragm and downwards into the pelvis, and large masses of granulation tissue filled with fungus may result. Deposits may be formed in the liver, infection probably going via the tributaries of the portal vein.

Thoracic actinomycosis usually attacks the bases of the lungs, extends into the subpleural connective-tissue plane, and spreads backwards or forwards along the diaphragm, softening, and causing an abscess which points somewhere along the line of attachment of that muscle.

Extension into the paravertebral groove may occur. Severe pain may be felt along the course of the intercostal nerves.

When actinomycosis gains access by accidental direct inoculation through the *skin* the lesion may not differ from an ordinary septic wound except that it is more chronic, tends to heal up in one place and break out in another, and is but slightly if at all painful.

Diagnosis.—The lesions caused by actinomycosis must be distinguished from other chronic inflammatory conditions and from new growths. Chronic sepsis, tubercle, syphilis, and sarcoma must always be considered. In the cervico-facial region actinomycosis frequently starts more acutely and causes a swelling which resembles that due to a *parotitis*; as the condition becomes more chronic its characters become more typical. Actinomycosis develops more quickly than *tubercle*, and whilst lymphatic glands are commonly affected by the latter, they are not enlarged by the former condition. Tubercle does not cause the formation of such hard masses of tissue, and when an abscess forms the pus does not contain the little granules. *Syphilitic lesions* are generally quicker in their development, and do not often attack the parts which are the common sites for actinomycosis. The Wassermann reaction should be positive in cases of syphilis, and treatment by salvarsan or by mercury and potassium iodide should soon cause decided improvement. If the lesion of syphilis breaks down and forms an ulcer with a "wash-leather" base, diagnosis is easy. For *septic lesions* an obvious cause can generally be found, and pus-formation is more rapid. There are cases, however, in which the septic process is chronic and forms much indurated tissue, and may give rise to considerable difficulty in diagnosis. This is increased by the common occurrence of secondary infection. A *sarcoma* progresses steadily and never recedes in one part while advancing in another. No pus containing the fungus granules is formed. In the jaw region a radiogram is very useful in helping to differentiate.

It may be stated definitely that any very hard, painless, chronic smooth swelling in the neighbourhood of the angle of the jaw is more likely to be due to actinomycosis than to anything else. The only absolute scientific proof is the discovery of the fungus in the discharge or in microscopic sections of the tissue. Nevertheless, clinical signs and symptoms are sometimes more helpful than pathological investigations, for infection with secondary organisms

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often masks the presence of the streptothrix; moreover, it is often months before any discharge appears, and frequently it is very difficult to find any fungus in sections of the tissue.

Thoracic actinomycosis may simulate *sub-phrenic abscess* or *basal tuberculous infection*. Diagnosis is made either by discovering the fungus in the sputum, or more usually by investigating the contents of the abscess, which points through the chest-wall sooner or later.

The appendiculo-cæcal type is not generally diagnosed until a persistent indurated sinus after appendicectomy (or the drainage of an appendix abscess) calls attention to something abnormal. Scrapings of the sinus will show the fungus. The type which develops insidiously will only be recognized when the swelling is explored and the pus examined.

Prognosis.—A fair number of recoveries take place when the infection is in the cervico-facial region. Thoracic actinomycosis is usually fatal, although the disease may last for many months. The cæcal type is also of bad prognosis, though in young people the chances are more favourable. As with tubercle, the prognosis is worse with advancing age.

Treatment.—Actinomycosis may be attacked by drugs administered internally, by surgical intervention, or by vaccine-therapy. The only drug which appears to have any influence is potassium iodide, which should be administered in maximum doses; but it is by no means a specific. Salvarsan, however, has been of benefit in some cases. Surgical measures are necessary to provide drainage when required, and in some cases to excise as much of the mass as possible. Indolent sinuses also need to be scraped. Either a stock or preferably an autogenous actinomycotic vaccine should be administered. The doses should be small at first, and later can be regulated to a certain extent by the reaction. Any secondary infection should also be treated by the appropriate vaccine. Attention to the general health is imperative.

ZACHARY COPE.

ACUTE YELLOW ATROPHY (*see* LIVER, ACUTE YELLOW ATROPHY OF).

ADDISON'S DISEASE (*see* SUPRARENAL GLANDS).

ADENITIS (*see* LYMPHATIC GLANDS, ENLARGEMENTS OF).

ADENOIDS.—Overgrowth of the adenoid tissue of the naso-pharynx.

Etiology.—Adenoids may be congenital in origin or may develop during the first few months or years of the patient's life. It is not unusual to meet with cases in which the symptoms due to the growth come on quite rapidly; in these a history of frequent colds or an attack of measles or scarlet fever can usually be obtained, and it is probable that a small mass was present from birth and that the causes mentioned resulted in a rapid increase in its size—a view often supported by a history of carache or of difficulty in taking the breast or the bottle in early life. Decrease in the size of the mass usually takes place at the age of puberty, but it never disappears entirely and its presence is always a source of danger owing to the secondary aural or general diseases which it may produce. Both sexes are equally liable to adenoids.

Symptomatology.—Nasal obstruction and frequent colds are the commonest symptoms. Snoring at night occurs in the large majority of cases, and during infancy there is often difficulty in taking the breast or the bottle. The child is usually under-developed and mentally dull (*aproseria*), its appearance suggesting that its intelligence is not of a high level. The nostrils are narrow and the palate is arched in most cases, and deformity and under-development of the chest are frequently seen (*see also* NASO-PHARYNGEAL OBSTRUCTION). Repeated attacks of bronchitis occur in most cases, sooner or later, owing to the mouth-breathing necessitated by the presence of the mass, to which should probably be ascribed also the dental caries that is usually in evidence. In some cases loss of smell, night terrors, paroxysmal cough, and incontinence of urine and even feces are present. Secondary symptoms, such as acute and chronic middle-ear suppuration, deafness from obstruction of the Eustachian tube, laryngeal spasm, enlarged cervical lymphatic glands, acute and chronic laryngitis and bronchitis nearly always supervene sooner or later.

The mass can be detected in the postnasal space (*see* NOSE, EXAMINATION OF).

Treatment.—There is only one form of treatment—removal, which should be carried out in all cases, irrespective of the size of the growth and whether secondary symptoms are present or not. The effects of this operation upon the patient's general development and upon the secondary complications, such as middle-ear suppuration, deafness, and chronic bronchitis, are too well known to require

ADENOIDS AND TONSILS, REMOVAL OF

description. One very important procedure, however, must be emphasized, as its omission often nullifies completely the beneficial effects that should follow operative treatment: the patient's nose must be examined. It is far too often assumed that all the symptoms are due to the presence of the mass of adenoids, whereas, in quite a large percentage of cases, deflections of the septum, spurs or enlargement of the turbinal bones are also present; if these conditions are not corrected the patient's symptoms will only be relieved slightly, if, indeed, they are relieved at all.

The operation is described in the next article.

G. N. BIGGS.

ADENOIDS AND TONSILS, OPERATION FOR REMOVAL OF.

—Many different operations have been devised for the removal of tonsils by various forms of guillotines; it is not proposed to describe all these methods here, but it is necessary to say that many of them are good and in the hands of their exponents give excellent results. The chief cause which leads to failure is incomplete removal, the tonsil being merely sliced and not eradicated. Each case must be dealt with on its merits, for it is no more justifiable to dissect out every tonsil than to remove every tonsil by the guillotine. The methods for dissecting out the tonsil and for morcellement are not described here, for they both require great skill, and many serious accidents are likely to occur should the operation be carried out by those who have not had a large experience in these methods.

In selecting the procedure to be adopted, certain rules should be borne in mind:

1. The ordinary enlarged tonsils in children can quite satisfactorily be removed by the different methods of guillotining.

2. If there are enlarged cervical glands which are obviously tuberculous, or if there is the slightest suspicion of their being tuberculous, the tonsils should carefully be dissected out.

3. If it is impossible to guillotine the tonsils, owing to their being buried or adherent to the pillars of the fauces, they may either be removed by morcellement, with punch forceps, or dissected out.

The following guillotine operation is that which I usually employ and which has been found to give excellent results, but I am far from claiming that it is superior to any of the other methods. The practitioner will find that there is no one method which will give, without continual practice, perfect results, and that all

the methods, with this proviso, will prove to be equally good. Should he be accustomed to carry out some other method which he has found to be satisfactory, he is strongly advised to adhere to it. Continual practice is the factor which commands success in this regard.

Operation. Removal of tonsils.—The patient, after being anæsthetized, should be placed in the recumbent position with a pillow under the head to raise it slightly, and the mouth should be held open with a gag. The guillotine selected should always be one the ring of which looks just too small to fit the tonsil; its blade should not be too sharp. When introduced it should be threaded over the dependent portion of the tonsil first, then pushed outwards as far as possible and, before the blade is forced home, pulled slightly forwards. The anæsthetist should be asked to steady the head, and at the same time to push in the tonsil by pressing just below the angle of the jaw. Directly the blade has been pushed "home," the distal end of the instrument should be shot towards the mouth; this will prevent the tonsil from slipping into the larynx. The opposite tonsil should then be removed in the same way.

If the blade of the guillotine is too sharp, the tonsil may slip out of the ring of the instrument whilst it is being withdrawn and become impacted in the larynx, and tracheotomy may have to be performed to relieve the condition.

Removal of adenoids.—A caged adenoid curette should then be inserted on its side and pushed back until it is in contact with the posterior wall of the pharynx, thus preventing the teeth of the instrument from being caught in the uvula. It is now pushed up behind the soft palate by a turning movement of the hand, drawn slightly forwards and pushed upwards and backwards. It will now have engaged the mass of adenoid tissue, when a rapid downward sweep in the middle line will remove most of the mass, and the instrument should be withdrawn. A second curette without a cage should now be inserted, and any lateral portions of the growth which have escaped the caged instrument can rapidly be removed by brisk curetting. Finally, the finger should be inserted into the naso-pharynx to make certain that all the adenoid tissue has been removed and that no "tags" remain.

After the tonsils, or the adenoids, or both, have been removed, the patient should be turned on to one side and his face bathed with ice-cold water. Hæmorrhage ceases rapidly, as a rule, and rarely calls for such

ADENOMA SEBACEUM

methods as plugging the postnasal space or stitching the pillars of the fauces. When it is excessive it is usually due to adenoid "tags," the removal of which generally stops it quickly. If there is general oozing from the tonsillar bed, pressure with a gauze plug for a few minutes usually arrests it. If a bleeding vessel can be seen it should be caught with forceps and twisted or ligatured.

Care must be taken not to injure the palate. This accident is usually due to the curette being pushed upwards and backwards in an endeavour to engage the adenoid mass before the instrument has slipped behind the soft palate, or to its not being inserted on its side, so that the hooks are apt to catch in the uvula, with disastrous results.

After-treatment.—The patient should be kept in bed for a few days, and should suck ice at intervals for the first twelve or twenty-four hours; this not only checks the bleeding but relieves the pain from which the patients sometimes suffer. Liquid nourishment and jellies should be given for the first two or three days, after which the patient may gradually return to a normal diet.

Secondary hæmorrhage rarely occurs, and is usually checked by sucking ice and by a hypodermic injection of morphia. Injections of horse-serum give excellent results in severe and persistent hæmorrhage. A mouth-wash of chlorate of potash (10 gr. to the ounce) should be used for a few days following the operation.

G. N. BRIGGS.

ADENOMA SEBACEUM.—A rare developmental anomaly, consisting of small tumours of sebaceous-gland origin, situated usually on the nose and cheeks.

Etiology and pathology.—The affection is congenital, although the lesions are not always present at the time of birth. They have been known to appear as late as the nineteenth year. The presence of the lesions should always lead the practitioner to inquire into the mental condition of the patient, as the majority of cases are associated with defective mental development. In most asylums for imbecile children cases are to be found. The disease is common among children of the poorer classes. In some cases the brain shows the condition known as tuberoses or nodular sclerosis, in which there are numerous nodules, consisting of proliferated neuroglia, embedded in the cerebral cortex and scattered over both hemispheres. The affection may also be associated with other

ADIPOSIS DOLOROSA

congenital tumours of the skin and internal organs. Microscopically, the findings in the skin vary. The majority of cases show a considerable hyperplasia of the sebaceous glands, but in some instances there is also a hyperplasia of sweat-glands, blood-vessels, or fibrous tissue.

Symptomatology.—Clinically, the disease is usually very typical. The lesions are small, varying in size from a pin-head to a split pea, rounded or flattened, and usually covering the nose and flush parts of the cheeks; they may, however, be present on the forehead and chin. They are closely set together, and usually symmetrical in distribution. They may be normal skin colour, or brownish or reddish. In some cases the associated telangiectasis is the most prominent feature. The skin covering the tumour is smooth or slightly warty. The lesions usually appear soon after birth, and grow gradually, with greater activity about the time of puberty. Some of the growths may disappear, leaving a slight superficial scar. Other cutaneous lesions, such as angiomas or fibromata, may also be present.

The condition causes no subjective symptoms and the **diagnosis** is usually easy. Although having the same distribution as rosacea, the early appearance and slow course of adenoma sebaceum easily distinguish it from that disease. The number of the lesions, their close grouping and distribution, distinguish it from other nævoid tumours such as benign cystic epithelioma.

Prognosis and treatment.—As a rule the condition is persistent, although spontaneous involution may occur. If removal of the tumours is desired, the best methods are electrolysis, the use of a fine-pointed thermocautery, and freezing for $\frac{1}{2}$ –1 minute with carbon-dioxide snow. Of these methods the third should be tried first.

R. CRANSTON LOW.

ADHERENT PERICARDIUM (see PERICARDITIS).

ADHERENT PLEURA (see PLEURISY).

ADIPOSIS DOLOROSA (*syn.* Dercum's Disease).—A rare malady, characterized by the presence of painful fatty deposits, together with asthenia and psychical phenomena.

Etiology and pathology.—This disease is of particular interest on account of the associated lesions in the thyroid gland, such as increase of connective tissue, dilatation of the

ADIPOSIS DOLOROSA

acini, and collections of round cells. The peripheral nervous system may also show microscopical evidence of disease. The malady is more common in women than in men, and tends to begin about middle life; it may also be encountered at quite an early age and in the old. Occasionally more than one member of a family is affected, and a history of alcoholic indulgence is frequent.

Symptoms and diagnosis.—It has been pointed out by McCarthy that this condition is not a disease *sui generis*, but one member of a group of allied conditions, all characterized by the formation of fatty masses which are not lipomata (see LIPOMATOSES). In one form—symmetrical adeno-lipomatosis—there are groups of tumours in the neck, axillæ, etc., while in adiposis cerebri, in which lesions of the pituitary gland are found, there is a general condition of adiposis and, usually, some defective development of the genitalia. The type known as adiposis dolorosa is characterized by the presence of fatty masses, which often begin insidiously as slightly reddened areas, forming a distinct tumour-like body, resembling to some extent a lipoma, but painful to pressure. Besides this local tenderness, spontaneous neuralgias are also experienced. Price, quoting Vitaut, describes three subtypes, the nodular, circumscribed diffuse, and general diffuse, but such a classification is unnecessary in view of what has already been said. A symmetrical arrangement of the masses is the rule, the lower limbs being especially attacked (Brocq), while the hands and face are usually exempt (Hyde). Most often the patients exhibit a considerable degree of asthenia, and flushings, purpura, hæmorrhages from the mucous membranes, cyanosis, anidrosis or hyperidrosis, are often met with. The knee-jerks may be absent, and arthritis and bullæ sometimes occur, evidence of profound trophic disturbance. There may be general obesity, and the patients are sluggish; occasionally they become insane.

Treatment is far from satisfactory. Thyroid extract should be administered; it has yielded good results in some cases. Baths and massage are helpful, while aspirin may relieve the neuralgic pains. Alcohol must be absolutely forbidden.

H. MACCORMAC.

AEROPHAGY (see FLATULENCE).

ÆSTIVO - AUTUMNAL FEVER (see MALARIA).

AGGLUTININS (see IMMUNITY).

ALCOHOLISM

AGORAPHOBIA (see PSYCHASTHENIA).

AGRAPHIA (see APHASIA).

AQUE (see MALARIA).

AIR SINUSES, ACCESSORY, DISEASES OF (see SINUSES, ACCESSORY. AIR, DISEASES OF).

AIR - PASSAGES, FOREIGN BODIES IN (see FOREIGN BODIES IN THE AIR-PASSAGES).

ALBINISM.—An absence or deficiency of pigment in the skin, the epidermal structures, and the iris and choroid. The condition is congenital and hereditary. The appearance of an albino is characteristic, the hair being straw-coloured, the skin very fair and delicately tinged with pink in the more vascular areas, such as the cheeks; the pupils red; and the irides pink. Since light enters the eyes through the unpigmented irides with abnormal intensity, the patient complains of much dazzling, especially in strong light, and nystagmus is commonly present. The only serviceable treatment is that directed to the eyes. Any defect of refraction should be corrected, and stenopaic glasses used for near work. Out of doors, tinted glasses may afford relief.

FREDERICK LANGMEAD.

ALBUMINURIA, ALBUMOSURIA, ALKAPTONURIA (see URINE, EXAMINATION OF).

ALCOHOLIC CIRRHOSIS (see LIVER, CIRRHOSIS OF).

ALCOHOLIC GASTRITIS (see GASTRITIS, CHRONIC).

ALCOHOLIC INSANITY (see ALCOHOLISM).

ALCOHOLIC NEURITIS (see MULTIPLE NEURITIS).

ALCOHOLISM.—Cases of alcoholic intoxication of the nervous system are consistently divided into the acute and the chronic, but it should be borne in mind that there is frequently a concurrence of the two, the chronic being constant and the acute episodal.

Acute alcoholism.—Among the acute forms ordinary drunkenness comes first in frequency and exhibits in its rapid progress that dissolution of the mind which is characteristic of

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chronic mental disorder. It is unnecessary here to detail the symptoms, but, as indicative of that which may be found among the more pronounced mental symptoms hereafter to be described, the reader may be reminded how differently alcohol affects different persons. Some become loquacious and some silent; some gay and some mournful; some quarrelsome and some affectionate; some foolish from the outset, and others, even up to an advanced stage, wiser and more witty than ever they are when sober. In the epileptic, drunkenness appears sometimes to be the exciting cause of a convulsive attack.

Delirium tremens.—Acute alcoholic delirium may occur as the result of a long drinking bout, the patient having previously been sober, but it is far more frequently an episode during the course of chronic alcoholic intoxication. In some instances of chronic alcoholism it occurs upon sudden abstinence from alcohol. It may also be excited in the chronically alcoholic by physical or mental shock or by illness—for instance, pneumonia. The most pronounced initial symptom is insomnia; this may be associated with physical restlessness and mental inquietude, headache, tremulousness, a furred tongue, constipation, and increased frequency of the pulse. During the course of a few hours or within a day or two, the patient becomes suspicious, and his mood is apt to alternate between passionate outbursts and gloomy depression. Hallucinations of sight or of hearing, as well as of the other senses, soon develop, and are fantastic and terrifying. Visions of wild and loathsome animals and of human enemies advancing to the attack with all sorts of weapons are very common. Mental confusion and disorientation are marked, so that the patient may have no correct notions of time and space or even of personal identity. Under the influence of the hallucinations he may be extremely restless, and may seek with all the strength of furor to combat his supposed assailants, or in terror may seek to escape them. In some cases epileptiform convulsions occur at this stage. Simultaneously with the mental symptoms the face is red and congested, the eyes are bright, the whole body is vibrating with tremor, the pulse-rate is increased, the temperature raised, and sleeplessness is absolute. As a rule, the symptoms abate in a few days, though they may be prolonged for a few weeks, and the tendency is to complete recovery. Other terminations are death in coma, or a condition in which hallucinations and delusions,

though of a much mitigated character, persist for an indefinite time.

Treatment of delirium tremens.—The patient should be put to bed and constantly attended in order that he may be prevented from injuring himself or others. Food is of great importance, and should be given in the most assimilable form whenever opportunity permits. Careful inquiries should be made as to what kind of alcohol, and how much, has been taken. The amount should be diminished on the first day by 2-4 oz., and this reduction should be continued from day to day so that in due course the patient gets no alcohol at all. During this time of progressive reduction hypnotics should be given; even if they fail to give sleep they tend to abate restlessness. In certain cases they seem to increase restlessness; they should not then be persisted with. Among the best are chloral hydrate and potassium bromide, of each of which 10 or 15 gr. may be given three times a day. Hyoscine hydrobromide $\frac{1}{100}$ gr. is useful as an occasional dose when the delirium is very intense. In sthenic cases large doses of calomel are valuable; in the enfeebled some less drastic aperient should be employed. Lumbar puncture and hot and cold packs may also be found useful.

Mania a potu.—Another group of acute mental symptoms excited by alcohol goes under this name. The affection generally occurs in those of bad family history who in their own persons exhibit signs of a neuropathic disposition. It consists in a state of intense excitement, with all the signs and symptoms of acute mania. It may occur in a predisposed person after the taking of an amount of alcohol absurdly small—for instance, a glass of wine—lasts from a few days to a few weeks, and ends in recovery. Treatment lies essentially in absolute abstinence from alcohol, and during an attack is precisely that employed in maniacal states.

Dipsomania.—This condition is only accidentally associated with alcohol, and should more properly, but not so conveniently, be placed with other commanding impulsions. It is a state in which from time to time the patient's volition is overborne by an imperative impulse to drink, just as in other cases the patient is impelled to the commission of some absurdity or crime. Between the dates of the occurrence of the impulse the patient is of perfectly sober habits, and may view with horror the periods of depravity to which his

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malady consigns him. Symptoms preceding the stage of the imperative impulse consist in failure of attention, irritability, depression, and loss of appetite. A drinking bout is then forced upon the patient by the perversion of his volition. When this is over, all is well until the impulse arises again. *Treatment* consists in endeavouring to get the patient to place himself under efficient protection when he feels the prodromal symptoms coming on. Suggestion is also of considerable value in lessening the imperious character of the morbid impulse.

Chronic alcoholism.—Among the manifestations of chronic alcoholism, it is first of all convenient to describe the condition of general mental deterioration which is associated with it, afterwards passing in review some of the more outstanding symptoms which have suggested a further subdivision into particular groups.

Alcoholic dementia.—When alcohol has been imbibed in quantities and over periods which vary enormously with different individuals, a time at length comes when symptoms of mental and physical deterioration make their appearance. Change of character and of business capacity are the first alterations to attract notice. Unpunctuality in time and in the performance of obligations, lack of attention, impairment of judgment and hesitation of volition, are succeeded sooner or later by irritability, depression, untrustworthiness, neglect of family and business, absurd quarrels and equally absurd friendships, offensive, scandalous, and obscene conduct, neglect of personal appearance and cleanliness, and loss of memory. The amnesia is very pronounced, and at first consists mostly of failure in the fixation of new impressions, but it rapidly increases and affects also the conservation of those that have been fixed in the past. With these mental symptoms there are sometimes tremor, altered facial aspect, altered reflexes, hyperæsthesiæ or hypoæsthesiæ, and evidences of affection of the alimentary, respiratory, circulatory, excretory, and nervous systems.

Dementia with delusions and hallucinations.—Engrafted upon this state of mental deterioration there may be delusions of suspicion, persecution, or grandeur, or a combination of these, as of jealousy. So also there may be hallucinations, and the patient may hear voices accusing or threatening him. Hallucinations of the other special senses, too, are not uncommon. *Treatment* lies in weaning the patient from alcohol. This is almost impossible unless

he can be placed in a proper institution where not only is alcohol withdrawn but the physical and mental states receive due attention. In these circumstances many patients improve enormously, but a residue remain permanently at a low mental level, with or without delusions and hallucinations.

Pseudo-general paralysis.—In some cases of alcoholic dementia there are such physical signs as slurring and hesitating speech, inequality and sluggishness of the pupils, and alteration of the character of the tendon reflexes, which, in conjunction with grandiose or other delusions, complete the clinical picture of general paralysis. Diagnosis is then best made by examination of the cerebro-spinal fluid, which in cases of alcoholism uncomplicated by syphilis shows no lymphocytosis and does not give a positive Wassermann reaction.

Korsakoff's syndrome.—Another group of symptoms consists in the combination of peripheral neuritis with mental confusion and disorientation in time and space, incapacity to acquire fresh impressions, loss of memory, and the fabrication of stories concerning the past, or pseudo-reminiscences. This is known as Korsakoff's syndrome, and is met with not only in cases of alcoholism but also in toxic states of other origin.

Treatment of alcoholism.—In all cases alike, treatment should be directed towards cutting off the patient's supply of alcohol, endeavouring to make good the bodily deterioration produced by it, and by various methods seeking to establish or to strengthen the patient's will not to take it in the future. It is very difficult to attain these ends until the patient has consented to enter a special institution, for it is but in very few cases that, without the assistance of some form of restraint, a successful appeal can be made to the patient's sense of propriety or to his reason. It must be remembered that such an appeal is not being made to one of normal mentality but to one whose affectivity, judgment, and volition are weakened; indeed in some cases to one who can scarcely be regarded as more than an automaton who will imbibe alcohol when in proximity to it much as a sponge soaks up water. Various drugs have been vaunted as of value, and to a greater extent by those who are not physicians than by those who are. We are content here to lay down the general propositions that alcohol should be withdrawn, and that such symptoms as the patient exhibits when withdrawal is being

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effected should be carefully attended to and met by the appropriate therapeutic measures. Psychotherapy is of vast importance, and may lie in suggestion or re-education. Every influence which can appeal to what remains of good in the patient's character should be employed to enable him to see the folly of his course, the reasonableness of sobriety, and the supreme desirability of self-control.

E. D. MACNAMARA.

ALEXIA (see APHASIA).

ALIMENTARY TOXÆMIA.—A toxæmia induced by the absorption from the alimentary canal of poisons resulting from bacterial activity. The term is lacking in scientific precision, since it implies the absorption of any poison from the alimentary canal; but, generally, it is not intended to include poisoning by drugs administered orally or rectally or by food. It is probable, besides, that in many patients said to be suffering from alimentary toxæmia an actual subinfection has taken place, the micro-organisms having gained an entrance into the blood from the alimentary canal.

Etiology.—The cause of the condition is obscure, and much more careful and prolonged investigation is required before any dogmatic views can be acceptable. It is clear that several possible factors must be considered. Unusual organisms may be present in the alimentary canal, or the normal flora may be exceptionally virulent or prolific. The latter is the view commonly held. A greater preponderance of organisms may be due to unsuitable food, to abnormal processes in digestion, or to the absence of protective bacteria, such as lactic-acid forming bacteria. The rate of absorption of the toxins and the state of the mucous membrane of the bowel probably play an important part, for it is more than likely that toxins are being constantly produced in the alimentary canal under normal conditions.

Undue retention of the contents of the bowel, a state to which the term "intestinal stasis" is applied, is the chief factor according to some authorities. The ill effects of constipation are well known, as is also the fact that some persons are much more susceptible to them than others. The supporters of the intestinal-stasis view, however, lay great stress on a mechanical obstruction of the bowel by peritoneal bands. As a result of the assumption of the erect position by man, there is supposed to be a tendency for the intestines to drop. To

counteract this the peritoneal bands are formed, and produce kinking and, consequently, stasis. Others hold that the peritoneal bands are not evolutionary but inflammatory, the result of bacterial invasion. Against the importance of the bands in this connexion, it is urged that they are present in the foetus, and that in conditions like tuberculous peritonitis, where they are in great profusion, the clinical picture of alimentary toxæmia is not met with. Moreover, these authors do not take into consideration the powerful effect of the ileal sphincter in regulating the flow of the contents into the large intestine. They regard the colon as a vestigial organ, devoid of physiological importance, and serving as an incubation chamber for deleterious organisms. This assumption cannot be said to be justified by inferences drawn from the evil effects of diseased colons. It is claimed that intestinal kinks and resulting stasis are demonstrable by radiography after the ingestion of bismuth meals, but before accepting this evidence more radiograms of normal subjects under similar conditions are required. Displacement of the bowel due to the weight of the bismuth cannot be ignored, nor must it be forgotten that the X-ray pictures have been taken in a single plane.

Another factor to which sufficient attention has not been paid is the normal defensive powers of the body against alimentary toxins. Toxæmia may conceivably be produced because the protective agencies of the intestinal mucosa, the liver, the thyroid, or the body fluids are defective.

Of the nature of the toxins themselves we are almost wholly ignorant. They may originate from dead bacteria, or they may be formed by bacterial action on the intestinal contents. Indol is known to be one of the products of bacterial putrefaction, and for many years was considered as the chief toxin of alimentary toxæmia. In this regard much work has been done on the excretion of indican and ethereal sulphates in the urine. Although, without doubt, there is often a relation between the excretion of these bodies and the symptoms ascribed to alimentary toxæmia, they may be present in the urine for years without symptoms, and are sometimes absent in patients believed to be suffering from that condition. It is no longer accepted that indol, indican, or ethereal sulphates are the toxins chiefly concerned. It is probable that the offending toxins may be comparatively simple chemical bodies derived from the proteins of the food.

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Tyrosin and histidine may be converted into poisonous substances, which, when absorbed, appear to be capable of producing arterio-sclerotic changes and, subsequently, fibrosis of the kidneys. Mellanby, Twort and others have shown that there exists in the intestines of animals a bacillus which is able to form from histidine a powerful poison, β -imidazole-thylamine. Work along these lines is yet in its infancy, and it must be confessed that the toxins of alimentary toxæmia have not yet been elucidated.

Clinical manifestations.—There are few morbid states which have not been ascribed to alimentary toxæmia by extremists, and their mere enumeration would need more space than the subject warrants, at any rate until our knowledge concerning it is more definite. The form which is least open to criticism is that known as *pyorrhœa alveolaris*, the local signs and treatment of which are described in the article on that subject. Under the name of "oral sepsis" this and other septic conditions of the mouth have attracted a great deal of attention in recent years, due, in great part, to the writings of William Hunter. The organisms from the septic teeth are said to infect the naso-pharynx and the whole of the alimentary canal. Thus there may arise tonsillitis of every degree and variety, pharyngitis, adenoids, septic gastritis, gastric ulcer, duodenal ulcer, enteritis, and colitis of every intensity, and even appendicitis. Infection of the glands leads to adenitis with its acknowledged predisposition to infection by tubercle bacilli. Infection of the blood may occur. Hunter describes also a severe form of anæmia resembling pernicious anæmia, which may occur alone or be engrafted on the anæmia of other diseases, is connected especially with septic gastritis, and, though severe, gradually disappears if the usual remedies are employed after the mouth has been attended to. Muscular pains and stiffness (fibrositis) and arthritis, particularly of the rheumatoid form, have also been ascribed to oral sepsis. This list of affections, although incomplete, gives an idea of the seriousness of oral sepsis as it is regarded by some authorities. There is no doubt that septic states of the mouth have been neglected in the past, but the tendency at the present time is to exaggerate their importance, and the wholesale removal of healthy teeth on the plea of oral sepsis cannot be too strongly condemned.

Turning now to intoxication from the bowel,

it will be agreed that constipation brings with it a fairly definite train of symptoms, which vary greatly in their severity in different subjects. While some experience considerable discomfort after constipation lasting only a few days, others remain in apparently good health, although evacuations occur as rarely as once a week or even at longer intervals. Headache, lassitude, a furred tongue, want of appetite, an unpleasant taste in the mouth, foul breath, nausea, vomiting, dyspeptic symptoms, slight jaundice, and depression of spirits are among the common results. Habitual constipation leads to a perpetuation of these symptoms, loss of fat may occur, the skin may become inelastic and pigmented, and there may arise considerable want of tone both of voluntary and involuntary muscle.

When considering other conditions which have been laid at the door of intestinal toxæmia we stand on less sure ground. It is well known that Metchnikoff believed that premature senility is explained in this way. Among other conditions ascribed to poisons derived from the bowel are arterio-sclerosis, cirrhosis of the liver, fibrosis of the kidneys, fibro-cystic disease of the breast, goitre (including exophthalmic goitre), arthritis (especially rheumatoid arthritis), myocardial atony and degeneration, presbyopia, cataract and other ocular diseases, microbic cyanosis, cyclical vomiting, and even mental disorders. Resistance to infections is said to be lowered. Cancer and tuberculous arthritis are included in the lists given by some authors. There is not sufficient evidence to prove that any one of these is definitely a result of alimentary toxæmia. We are still in the stage of conjecture merely, and much more work is necessary before the true can be winnowed from the false.

Treatment.—The essential treatment is the time-honoured procedure of keeping the bowels well open and so preventing the accumulation of noxious products and avoiding their absorption. The detailed treatment of constipation will be found under that heading. Pure liquid paraffin is especially useful in this condition. Treatment by intestinal antiseptics has not been found of great value in most cases, although their action is probably greater than has generally been supposed. For the more remote consequences of alimentary toxæmia, such as enlargement of the thyroid gland and rheumatoid arthritis, it has been claimed that they are beneficial, especially thymol in doses of 10 gr. and upwards, three times daily, for periods of

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two or three weeks. If thymol be given, fat must be excluded from the diet, and alcohol prohibited, since these act as solvents of the drug. Since lactic-acid-forming bacilli are inimical to the *Bacillus coli communis*, Metchnikoff introduced the "sour milk" treatment. The strain of organism which proved most reliable was the *Bacillus lacticus bulgaricus*, and cultures of this organism, either in the form of sour milk or in tabloid form, have been widely used. The results have not fulfilled the hopes which were at first entertained, and this form of treatment has now largely fallen into disuse. Although some cases seem to have been remarkably benefited by it, a few have certainly been made worse.

If the constipation is treated carefully and systematically, the need for the surgical measures which have been recommended, such as ileo-sigmoidostomy, colonic fixation, and colectomy, will rarely arise.

FREDERICK LANGMEAD.

ALOPECIA.—The generic name for baldness, whether partial or complete, congenital or acquired.

CONGENITAL ALOPECIA

Congenital alopecia is extremely rare. Several cases are on record where infants have been born with no hair, and where the deficiency has remained throughout life, but in most cases the baldness has been incomplete and a few patches of downy hair were usually present. In a certain number of cases the alopecia has been the result of delayed development, and the hair has grown later.

ACQUIRED ALOPECIA

The most prevalent types of acquired alopecia are the so-called *seborrhæic alopecia* and *alopecia areata*, but more or less complete baldness can occur also from a variety of different causes of which the following are the most important—

1. *Senile alopecia*.—In old age the hair-follicles and papillæ tend to atrophy, leading to a gradual falling of the hair, generally associated with whitening.

2. *Alopecia symptomatic of general diseases*.—Any constitutional disease which causes a lowering of the general vitality, such as measles, scarlet fever, typhoid, myxœdema, exophthalmic goitre, diabetes, pneumonia, and syphilis, may be responsible for thinning of the hair or even complete baldness, but this is generally of a temporary character.

3. *Alopecia due to nervous disturbances*.—General alopecia has been known to result from a severe nerve shock, while localized alopecia may be the result of injury or destruction of the nerve supplying the affected area.

4. *Alopecia dependent on morbid states of the scalp and hair-follicles*.—Any form of acute dermatitis, such as exfoliative dermatitis, acute eczema, or erysipelas, may be associated with temporary loss of the hair, while morbid conditions of the scalp leading to atrophy, such as lupus erythematosus, tertiary syphilis, and scleroderma, may result in the formation of permanent bald patches.

5. *Local irritants*, both chemical and parasitic, may cause temporary baldness; such are applications of cantharides or chrysarobin, or the action of septic micro-organisms or of the fungi of ringworm and favus. Applications of the X-rays also cause the hair to fall out, and if the dosage be excessive, permanent alopecia may result.

SEBORRHÆIC ALOPECIA

The most common form of baldness due to a morbid condition of the scalp is that known as seborrhæic alopecia. This familiar type begins on the vertex of the scalp and sides of the forehead, and gradually spreads till a large area on the crown of the head is involved. In it the scalp is usually greasy, and may be covered with fine powdery scales or "dandruff," or may present definitely inflamed patches covered with coarser waxy scales.

Etiology.—There is considerable variance of opinion as to the precise cause of this type of alopecia, but the prevalent view is that it is due to the action of certain specific micro-organisms growing on a soil rendered fertile by an excessive activity of the sebaceous glands of the scalp, a peculiarity of the person affected. It is occasionally met with in early life, about the age of 20, but is more common between 35 and 40, and is slowly and inevitably progressive. It affects males more than females, not infrequently occurs in families, and may be hereditary.

Prognosis and treatment.—Prognosis is unsatisfactory, but, if the affection is dealt with early, a good deal can be done by suitable treatment to retard its progress. As it is purely local, only local treatment is indicated.

In the oily type of case, where the scalp is greasy but not definitely scaly, it should be washed, on an average, once a week in men,

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and once a fortnight in women, with the following soap spirit lotion:—

- Ry Sap. virid. 2 parts.
Sp. vini rectific. 1 part.
Ol. lavand. q.s.

The soap should be rubbed into the scalp with a piece of flannel or sponge and rinsed out in warm water. As in women with long hair this procedure is apt to leave the hair sticky, the soap spirit may be mixed with warm water in the proportion of a tablespoonful to half a pint to form a shampooing lotion. Once or twice a day an antiseptic lotion such as the following should be thoroughly rubbed in:—

- Ry Acid. salicyl. gr. xxx.
Hydrarg. perchlor. gr. i.
Sp. vini rectific. ʒii.
Aq. ros. ad ʒvi.

In place of salicylic acid, resorcin is frequently employed, but should be used with caution in patients with fair or greyish hair, as it produces a rusty or sandy discoloration.

Where there is marked dandruff, greasy applications are more effective, and should be employed so long as the scaliness remains. In women a brilliantine such as the following should be rubbed in daily:—

- Ry Acid. salicyl. gr. x.
Ol. ricin. ℥xx.
Sp. vini rectific. ad ʒi.

In men an ointment may be substituted, such as—

- Ry Acid. salicyl. gr. x.
Sulphuris præcip. gr. xv.
Paraff. moll. alb. ad ʒi.

Where the scales are particularly thick and waxy, tar applications are of service, such as—

- Ry Resorcini gr. xx.
Ol. cadin. ʒi.
Vaselin ad ʒi.

As a routine procedure in moderately severe cases the brilliantine or ointment should be applied once a week the night before the scalp is washed, and the alcoholic lotion rubbed in daily.

ALOPECIA AREATA

In alopecia areata the baldness occurs in limited patches, but these may increase and coalesce until the whole cutaneous surface is involved. There are two main types, namely (1) a central type, the more common, in which various-sized patches are irregularly distributed over the central area of the scalp; and (2) a

marginal type in which the bald areas occur at the hairy margin of the scalp especially in the occipital and temporal regions. This division, however, is somewhat arbitrary, and many of the cases are mixed.

Symptomatology.—The affection usually begins on the scalp as one or more bald patches, but it may occur also in the beard, moustache, or eyebrows, and occasionally in any other part of the hairy system. As a rule the patch when first noticed is completely bald, but if seen earlier a few short hairs or stumps can generally be detected at the spreading edges. These stumps have a peculiar “point-of-exclamation” appearance when extracted, owing to the intrafollicular portion being thin and atrophied, while the free end is thicker and may be broken off and frayed out like a brush. At first also the patches may be slightly inflamed and oedematous. Older patches present an ivory-like whitening of the skin, may be smooth and glossy like a billiard ball, and are occasionally slightly depressed. In certain cases abnormal sensations in the affected areas may precede the defluvium of the hair by a few days, such as a feeling of heat or cold, tingling or definite neuralgic pain, and tenderness on pressure which may persist for some time after the hairs have fallen out. As a rule the patches heal spontaneously, and new hairs begin to grow in from a few weeks to several months. The new hair comes as a fine down, which on reaching a certain size is shed and replaced by stronger hairs similar to those of the rest of the scalp, but often lighter in colour, and sometimes actually white. In the course of a few months, however, they usually acquire the original tint. The affection has a marked tendency to recur, and subsequent attacks are, as a rule, more extensive than their predecessors. In universal cases not only the long hairs but the fine lanugo hairs of the glabrous skin may be involved, the hair falling out with extraordinary rapidity and perhaps being completely lost in the course of a few weeks. In the majority of these rapid and extensive cases there is a history of previous attacks increasing in severity.

Etiology.—The affection is most common in childhood and early adult life, and gradually diminishes in frequency after 30. It is equally prevalent in either sex. Its exact etiology is still under discussion, there being three main views with regard to its causation, namely, the microbic, toxic, and tropho-neurotic theories.

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According to the *microbic* theory the disease is due to some microbe or fungus situated about the hair-follicle which interferes with the growth of the hair. Up to the present time, however, there has been no micro-organism consistently found, nor is there any evidence that the disease is contagious or transmissible by inoculation. The supporters of the *toxic* theory explain the affection as the result of the action of a toxin reaching the hair papillæ by the blood-stream or locally produced in and around the hair-follicles. But the theory which has attracted most attention and is most generally accepted is the *tropho-neurotic*, according to which the fall of the hair is caused by an inhibition of the functions of the hair papillæ by disease or destruction of the nerve supplying them. In support of this view are cited the cases of universal alopecia which have occurred as the result of severe mental disturbances and those of localized bald patches which have resulted directly from injury of the nerve supplying the affected area, and have been associated with more or less severe neuritis. Where the bald patches have occurred in areas other than those supplied by the affected nerves, the defluvium has been ascribed to reflex action.

The **prognosis** in alopecia areata varies greatly in different cases, being dependent on the extent of the baldness, the rapidity with which the hair falls out, the number of previous attacks, and the age of the patient. In early attacks the prognosis is good, and a regrowth may be expected in from three to six months, while in later, more extensive outbreaks the outlook becomes increasingly unfavourable. In generalized cases the prognosis is always serious, and the hair may never grow again. If it has not begun to appear in a year it generally means that the baldness will be permanent, though cases are on record in which the regrowth has occurred several years after the hair fell out.

Treatment.—The treatment of alopecia areata is principally local, but to obtain the best results this should be combined with suitable *general measures*. Any defect of the health which may be even remotely connected with it, such as debility, mental depression, headaches, neurasthenia, etc., should be suitably dealt with. The hygiene of the mouth should be attended to, defective teeth removed or stopped, errors of refraction corrected, catarrh of the auditory canal treated, and adenoids removed, as any of these local conditions may reflexly influence the nerves of

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the scalp and be factors in the causation of the baldness. With regard to *local treatment*, it is of the first importance to attend to the proper hygiene of the scalp, and to remove dandruff and greasiness in the manner described above in connexion with seborrhœic alopecia. Those who believe in a parasitic origin of the disease advise the application of antiseptic lotions or ointments containing salicylic acid, mercury, sulphur, etc. More valuable, however, is the employment of irritant applications to improve the circulation and stimulate the hair-growth. At one time the customary treatment was blistering by the application of acetic acid, cantharides, etc., but equally good results can be obtained by less drastic measures and the maintenance of a mild degree of inflammation by painting the bald patches daily with linimentum terebinthinæ aceticum, with or without the addition of 1-3 per cent. of acetum cantharidis.

Various physical remedies have been recommended also, of which the most valuable are systematic massage of the scalp and the application of the actinic rays of light from a Finsen or a mercury vapour lamp.

J. M. H. MacLEOD.

AMAUROSIS.—This term (*ἀμαρῶσις*, to darken) has remained in use from pre-ophthalmoscopic days. Originally "amaurosis" meant a condition of blindness without obvious external changes in the eye. With the discovery of the ophthalmoscope the connotation of the name became much more limited, but it is still of value in denoting certain types of blindness, especially those not associated with obvious ophthalmoscopic changes. It is not possible, however, to give an accurate definition of amaurosis, the word still being used somewhat loosely. For example, an eye blinded by acute glaucoma or persistent irido-cyclitis is sometimes spoken of as amaurotic. Attacks of temporary amaurosis occur in conditions likely to give rise to arterial spasm, and are often probable indications of early arterio-sclerosis. One of the most interesting forms occurs in poisoning by quinine or by *Aspidium filix-mas*, in which the blindness is associated with intense spasm of the arteries. In quinine poisoning there is probably in addition a direct toxic effect on the ganglion cells of the retina.

In *uræmia* a sudden or rapidly developing amaurosis may set in without any obvious signs of retinitis. It is usually associated with

increasing headache, giddiness, and sickness. If the uræmic attack passes off, the sight may be restored to normal in a few days.

Similarly, in *cerebral tumours* attacks of temporary amaurosis, usually of a very fleeting character, occur, associated with a distinct increase of headache, giddiness, and sickness; it often lasts only for a minute or two, and may occur in cases where there is no papilloedema. It is probably due to the distended third ventricle pressing on the chiasma.

The term amaurosis is still applied to blindness resulting from diseases of the central nervous system, even though there may be obvious signs of optic atrophy. *Tabetic atrophy* is sometimes called spinal amaurosis, and many of the cases of blindness which we now know to be due to *pituitary growths* and other intracranial conditions are spoken of as cerebral amaurosis.

Complete monocular amaurosis is very rare as a functional manifestation, though in some cases of *hysteria* the amblyopia may approach to amaurosis. When complete amaurosis is complained of, the suspicion of malingering is at once aroused.

LESLIE PATON.

AMAUROTIC FAMILY IDIOCY (see IDIOCY, AMAUROTIC FAMILY).

AMBLYOPIA.—The term amblyopia (ἀμβλῦσις, dulled; ὤψ, eye) is a relic of pre-ophthalmoscopic days. As knowledge has advanced it has lost its original signification, and its present use is not easily defined. It is generally applied to defective vision not due to errors of refraction and not associated with obvious lesions of the eye.

The most important form of amblyopia is that which occurs in one eye as a result of *non-use* in the early years of life, either owing to an interference with the media that prevents the development of macular vision, or to suppression in the case of a squinting eye. If the cause is removed and training of the amblyopic eye can be undertaken at an early enough age, a very great improvement will often result, but after childhood little if any improvement can be hoped for. This condition is known as *amblyopia ex anopsia*. There are also cases of *congenital amblyopia* in one or both eyes, without any history of squint or interference with the media; when both eyes are affected, nystagmus is likely to be associated with the defective vision.

The *toxic amblyopias* form another important

group. Some of those of acute onset are referred to under AMAUROSIS, e.g. the blindness due to quinine and *Aspidium filix-mas* poisoning. *Tobacco* is the most frequent cause of toxic amblyopia. It is mostly the heavier and darker tobaccos, such as shag and black cavendish, which produce the condition, and there is very frequently an associated habit of spirit-drinking. Very often the precipitating cause of an attack of tobacco blindness is financial or domestic worry. Vision is not always equally affected in both eyes. The most important symptom is the loss or diminution of central colour vision, especially the perception of red and green. The primary seat of action of the poison is probably the ganglion-cell layer of the retina, and the changes in the retrobulbar portion of the nerve are probably in the nature of secondary degenerations. Other poisons, such as methyl alcohol, carbon disulphide, and even tea may produce similar effects. The *treatment* consists in cutting off absolutely the supply of the poison. Drinking large quantities of tepid water regularly is beneficial, especially if potassium iodide is also given. Strychnine, though not a curative agent, is of use in improving the conductivity of the nerve and so benefiting the sight while the poison is in process of reabsorption from the tissues. The patient's sight often deteriorates a little more in the first fortnight after giving up tobacco, and no great improvement should be expected till the end of six or eight weeks. At the end of six months sight should be almost normal.

Functional amblyopia may occur both in neurasthenia and in hysteria. The visual fields in such cases are of value when properly used and interpreted. In true neurasthenia one can undoubtedly obtain the spiral field as the patient tires. In hysteria it is possible to get almost any type of field and almost any form of inversion of the colour fields.

LESLIE PATON.

AMENORRHEA.—The absence of a menstrual discharge is readily classified under two headings: *primary amenorrhœa*, in which the menstrual flow has never been established, and *secondary amenorrhœa*, in which, once having been established, the menses for some reason fail to reappear over a longer or shorter time. It is to be remembered that amenorrhœa may indicate either inability of menstrual fluid to escape, or failure to produce such a fluid. Amenorrhœa is a normal condition at certain

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age-periods, and also during pregnancy and full lactation.

PRIMARY AMENORRHOEA

This is best considered under the two divisions of constitutional and developmental, which express broadly the origin and cause.

Constitutional primary amenorrhœa is found in anæmic girls working long hours in an unhealthy atmosphere, in chlorosis, and in extensive tuberculous disease. Any factor, acting either mentally or physically, which tends to overstrain the vitality of the already highly tried system at this particularly important epoch may cause postponement of the menstrual function. The history in these cases is important; the age, the conditions of life, at home, at work, or at school, symptoms suggestive of anæmia, constipation, signs of extensive disease, and the general state of development, should all be considered and may indicate the cause. There is, as a rule, complete absence of symptoms such as periodic malaise, with discomfort in the lower abdomen, and the slight general disturbance accompanying certain forms of secondary and developmental amenorrhœa.

Unless the patient has passed the average age at which menstruation begins (15 years) by two or three years, and unless there are symptoms pointing to retention of menses, or for some reason an examination is specially requested, it is unnecessary to make a complete examination. Palpation of the abdomen will eliminate an abdominal swelling, and evidences of attainment of puberty will be noted in the degree of development of the mammæ, the growth of pubic hair, and the correct formation of the external genitalia.

The **treatment** is directed to the causal factors, and, although the amenorrhœa may be the outstanding symptom, nothing in the nature of a specific remedy or of local applications is called for. The more difficult cases to treat are those in which the shop, factory, or school life is a source of overstrain, for change of work and new occupations are undertaken with great reluctance owing to the risk involved under modern competitive conditions. It will therefore be found that much thought is required when correcting errors in hygiene and drawing up rules for better health; it is often impossible for the patient to obtain the requisite open-air existence and exercise.

Developmental primary amenorrhœa occurs in two clinically distinct classes—one

in which the menses are retained, the other in which the menstrual fluid is not produced.

The various developmental errors which it is necessary to consider are tabulated for conciseness, and will be discussed in detail under these headings:

1. Ovaries, absent or deficient.
2. Uterus, (i) absent or rudimentary;
(ii) foetal and infantile.
3. Vagina, (i) absent;
(ii) atresia.
4. Hymen, imperforate.

1. **OVARIES.**—Absence and deficiency of these organs are both rare, and the defect cannot be verified during life. The **clinical features** of these cases are the absence of puberty changes, menstrual flow, and pubic hair, and a male type of figure.

2. **UTERUS.**—*Complete absence* is exceedingly rare.

The term *rudimentary* usually indicates a uterus represented by a fibro-muscular cord, frequently solid, but possibly containing a cavity in part of or throughout its length. The clinical features are modified by the ovarian conditions: in the absence of ovarian activity, female characters are in abeyance; when the ovaries are active, sex characters are normal and sexual desire is usually active.

The *foetal uterus* is characterized by the presence of a relatively large and typically thick-walled cervix, on the top of which there is a soft, rounded, small corpus uteri. With the sound the cavity measures 1–1½ in. The whole structure is smaller than normal and the cervix as a rule conical.

A greater degree of development with a more typically formed corpus uteri, the cavity of which slightly exceeds 1½ in. in length, is correctly designated the *infantile* uterus. The symptoms in each case are similar, and the two terms are frequently used indiscriminately.

Clinically, while amenorrhœa is the rule, a scanty flow with dysmenorrhœa may occur, in which case the periods are irregular. Sexual desire is lacking in many instances, and sterility is almost invariable. In many cases in which menstruation occurs the patient seeks advice on account of the dysmenorrhœa; others after a barren marriage desire treatment for the sterility.

The **diagnosis** may be exceedingly difficult. The perfect general development, the presence of sexual desire, regularity in the menses even though associated with dysmenorrhœa, will in many instances cause the uterine condition to

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be overlooked. In other cases, in which menstruation is in abeyance or irregular, the condition is more readily suspected. The confirmation is made by finding, on bimanual examination, the large, solid, conical cervix readily palpable, and the uterine body either insignificant, soft, and rounded, or at any rate smaller than the size of the cervix would suggest.

Treatment.—Many attempts have been made to induce a periodic discharge in cases in which menstruation is lacking. The measures that have been tried include glycerin and ichthyl tampons accompanied by hot sitz-baths, and, in the same category, passive hyperæmia, by applying a glass speculum over the cervix to act as a Bier's cup; this has been applied for ten minutes and repeated at regular intervals. The result has been in some cases temporary oozing, but never the establishment of regular menstruation. Again, many cases, on the supposition that the faulty uterine development is an indication of ovarian insufficiency, have been treated with ovarian extract, and also with thyroid, suprarenal, and pituitary-gland preparations, but with negative results.

The majority of these patients will at one time or another have been treated by drugs, local applications, and operative measures. Few escape dilatation and curettage, as, told by one surgeon that sterility is incurable, they are always ready to undergo further treatment at the hands of another.

3. VAGINA.—Complete absence is a very rare anomaly. Less rarely the vagina is represented throughout its length by a fibrous cord. The amenorrhœa may bring such patients to the doctor, but infrequently it is after marriage, when cohabitation has been found impossible, that advice is sought. The question will then arise whether treatment is to be attempted or the condition left alone. Often the sense of imperfection causes mental distress and morbid depression to an astonishing degree. The correct attitude would appear to be for those who anticipate marriage, or are actually married, to have laid before them the possibilities in the way of artificial-vagina formation, for which Baldwin's procedure is undoubtedly the best.

Atresia vaginæ is a term applied to any obliteration of the lumen of the vagina. In its simplest and more common form a thin membranous occlusion is found at the lower end. Almost the whole of the passage may be obliterated; if so, there is usually an

associated absence of uterus and Fallopian tubes.

Clinically, there is complete amenorrhœa, and the cases are divisible into two distinct classes—(a) those in which menstruation does not take place, (b) those in which the menses formed are retained.

(a) When no menstruation occurs, the only symptom is, as a rule, the amenorrhœa, unless, as occasionally happens, there are pelvic discomfort and general malaise at fixed intervals.

(b) If the menses are retained the symptoms are very similar to those of imperforate hymen (see below). In lesions involving the middle of the vagina, the fluid accumulates in the upper part of the vagina (*hæmatocolpos*) before it begins to exert pressure upon the uterus. It is not uncommon for the uterus and Fallopian tubes to escape distension, as the degree to which the vagina will enlarge is very considerable, and an appreciable swelling, rising in the hypogastrium, adds to the chance of early detection.

The **diagnosis** of atresia vaginæ is made from the history of amenorrhœa and of pelvic pain recurring at intervals of one month, and the discovery of a hypogastric tumour. The character of this tumour is ordinarily spherical, anchored, and slightly tender. Confirmation of the diagnosis must be made by careful bimanual examination, for which an anæsthetic will in most cases be required, when the presence or absence of a perineal swelling and the position of the occluding vaginal lesion will be determined. It is important also to make a rectal examination and to sound the bladder in order to learn the extent of the lesion in the vagina, because the treatment adopted depends upon the degree and position of the atresia.

Treatment.—In the presence of a perineal or vulval swelling and a simple occluding membrane to the vagina, the contents are simply evacuated. When, however, the occlusion is of greater extent, it is necessary to make a careful dissection between the bladder and the rectum in the line of the vagina until the swelling is reached, when a perforation into it is made.

If the lesion of the vagina affects the upper end, the question of removing the hæmatometra by the abdominal route presents itself. If only a small part of the vault is obliterated, a dissection may safely be made up to the hæmatometra.

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4. **IMPERFORATE HYMEN.**—In this condition there is a thin, firm membrane occluding the ostium vaginae. The retained menstrual fluid distends the vagina until it may contain some pints of fluid, and, as the distension increases, the mass tends to protrude at the vulva. Later, in large accumulations, an abdominal tumour is formed, readily palpable in the hypogastrium, and even extending up to the umbilicus. With the increase of size of the pelvic tumour there is usually pressure on the rectum. As the vagina dilates, and the hæmatocolpos rises in the abdomen, it lifts the bladder and causes considerable elongation of the urethra. In time the pressure of the vaginal fluid may cause dilatation of the cervix, and even of the uterus and tubes.

Clinically, this condition is associated with amenorrhœa, and mild periodic attacks of pelvic discomfort at intervals of one month. The amount of pelvic discomfort is often insignificant, and seems to depend upon the rate of accumulation. The physical signs are those of an elastic, spherical swelling, on some part of which a more solid mass representing the uterus may be encountered. On inspection of the external genitalia a swelling may at once be detected protruding between the labia—it is characterized by the bluish, congested, bulging membrane covering the protruding point. A finger introduced into the rectum encounters a large fluctuating swelling, practically blocking the pelvis.

Treatment.—The fluid should be evacuated. The operation is a simple one, but the risks of infection are considerable. An incision is made through the dark-red cap of the hæmatocolpos; the fluid drains away slowly, owing to its viscid, "treacly" character. When the vagina appears to be empty it should be explored with the finger and the condition of the cervix, uterus, and appendages determined.

It is well to excise the obstructing membrane and to suture the edge of the vagina to the skin-edge all round, otherwise the aperture in the hymen tends to close, and the frequent use of bougies may be necessary to keep it patent. When the uterus is distended its contents will drain away with the vaginal fluid.

SECONDARY AMENORRHEA

Etiology.—Secondary amenorrhœa may be due to general or local conditions.

The general causes include such maladies as anæmia, chlorosis, malaria, Bright's disease, diabetes, acute fevers, cachectic states, and

tubercle. In these conditions it is but a minor symptom, but in many anæmic girls it provides the chief anxiety. The occurrence of amenorrhœa in some cases of chlorosis has never been explained, but the fact that in other instances there may be menorrhagia suggests that the explanation lies in an altered metabolism and a lowered level of vitality. The blood should in all cases be examined to determine the type of anæmia. The cause is as a rule obvious, and the treatment required is directed to improving the general health.

Obesity is usually given as a common cause of secondary amenorrhœa. The obesity is perhaps more correctly to be regarded in the majority of cases as an indication of ovarian insufficiency. The typical case of this type is found in big, large-framed women in whom menstruation became irregularly established with scanty loss and long periods of amenorrhœa. The patients are usually well formed, and as the periods become less the deposition of fat often increases. These cases seldom improve, but occasionally a long course of thyroid extract, using small doses at weekly intervals, is beneficial.

Among the *toxic* causes are addiction to drugs such as morphia and alcohol, and lead-poisoning, which often cause amenorrhœa.

Of *nervous origin* may be instanced the cases following severe fright, although the same factor has been followed equally often by excessive loss. The delayed period often seen in women who have cause to fear a pregnancy, and pseudocyesis, indicate the same influence in operation. Pseudocyesis is detected by noting the expiratory position of the chest, the prominent doughy abdomen, the refusal of the patient to respire abdominally, and the absence of uterine enlargement. Psychic disturbances, especially melancholia, are commonly associated with suppression of the menses. Occasionally, after marriage, amenorrhœa which is not physiological is noted.

Local conditions causing secondary amenorrhœa are numerous, and many of them obscure.

Ovarian insufficiency.—The following may be regarded as instances of ovarian insufficiency: In myxœdema, amenorrhœa is a constant symptom and is invariably corrected by the administration of thyroid extract. In the later stages of exophthalmic goitre the function is also in abeyance. In pituitary lesions such as acromegaly, amenorrhœa may occur, and again with diminished pituitrin secretion the same phenomenon is noted. In true

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hermaphroditism, with the advent of function in the testicle, male characters predominate and the menses are suppressed.

The menopause sometimes takes place prematurely. In such cases, after a variable period of normal life, the menses suddenly cease, and then follow all the typical symptoms of the climacteric. This menopause occasionally follows a first or even a second pregnancy.

An artificial menopause is seen after removal of both ovaries by surgical means, or with extensive growths of both ovaries. In the case of ovarian new growths it is remarkable to what size they may attain before destroying completely the ovarian tissue and so affecting menstruation; on a tumour the size of a full-term gestation sac a corpus luteum may be encountered.

An artificial menopause is now not infrequently seen as the result of Röntgen radiation applied in the treatment of myomata of the uterus. As the most direct and immediate result is upon the ovary, in which atrophy occurs, amenorrhœa is the rule, while the severity of the climacteric symptoms varies considerably. With radium, especially as applied to the interior of the uterus, the effect upon the ovaries is not so immediate, although an artificial menopause is often induced.

Uterine and vaginal causes. An atrophy is occasionally found to occur. It may represent a condition of superinvolution, or be the result of prolonged lactation. In some cases, following a pregnancy, the menses fail to reappear; in other cases, after as many as five to seven years of amenorrhœa subsequent to a pregnancy or prolonged lactation, menstruation may recur regularly, or a patient may forthwith become pregnant. There is no treatment beyond that directed to favouring general good health.

Acquired atresia of the uterine body, the cervix, or the vagina may occur. It rarely affects the body of the uterus, but is found as the result of tuberculous lesions. In the cervix the occlusion is usually subsequent to the application of caustics. When occurring in the vagina it is often incomplete and follows sloughing from injury during parturition. When acquired atresia of the uterine body or cervix occurs, there is an early formation of hæmatosalpinx with the associated amenorrhœa. With occlusion of the vagina, a careful examination will be necessary to determine the extent of the lesion. It is often

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advisable to sacrifice the uterus, as the scar tissue renders dissection between the bladder and rectum exceedingly difficult.

Treatment of amenorrhœa in general.

—There are many drugs supposed to induce menstruation, but their efficiency has often been questioned. It is well before using them to eliminate general constitutional causes, such as anæmia, chlorosis, and depressed vitality.

In chlorosis, with suitable dietary, purgation, iron and arsenic medication, the menses will return.

In cases where the general vitality is lowered by overwork it is sufficient to alter the mode of life, insisting on a certain amount of healthy exercise and fresh air, while the diet is simple and the bowels are made to act regularly.

In cases where there is no obvious fault of development and no constitutional cause, such drugs as rue, savin, and saffron have been extensively employed, but with doubtful result. More popular is the active principle of parsley, known as apiol (dose 2-5 min.), frequently administered in combination with ergot. Manganese salts have also been used.

In cases associated with rapidly increasing obesity, dieting, baths, and exercise, together with the administration of thyroid extract and also of ovarian extract (the latter very disappointing in its action), should be tried.

Electrical treatment formerly enjoyed a certain reputation, but is not to be recommended, the results scarcely justifying its use.

In cases where symptoms suggestive of menstruation recur periodically, a hot bath may be advised on the first day, or a day or two previously on the next occasion; it may be successful in inducing a flow.

BRYDEN GLENDING.

AMENTIA (*see* IDIOCY).

AMNESIA (*see* MEMORY, DISTURBANCES OF).

AMŒBIC DYSENTERY (*see* DYSENTERY).

AMYLOID DISEASE (*syn.* Lardaceous Disease; Waxy Degeneration).—A degeneration of connective-tissue fibres characterized by the formation of an albuminous substance known as lardacein.

Etiology.—The chief causes of amyloid degeneration are chronic suppuration, syphilis in the later secondary and tertiary stages, and chronic tuberculosis. The forms of tuberculosis which lead to it are those in which a secondary infection has occurred, such as

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phthisical cavities, and chronic joint-disease or spinal caries associated with discharging sinuses. It has also been described in rheumatism, leukaemia, lymphadenoma, chronic malaria, chronic dysentery, lead-poisoning, and cancer.

Pathology.—The disease is essentially a degeneration of connective-tissue fibres. It affects first the delicate connective tissue between the muscle-fibres of the smallest arterioles. Peripherally it spreads to the perithelial supports of the capillaries and the connective tissue in the veins. Centrally it spreads to the coats of the larger arteries. Exceptionally its distribution is otherwise, as in the diffuse form of amyloid degeneration of the spleen, where it begins in the walls of the venous sinuses and in the reticulum of the spleen pulp. The degeneration is accompanied by considerable swelling of the connective tissue, so that neighbouring structures are pressed upon, and may atrophy and be rendered functionless. Thus, destruction of the muscular fibres of the arteries interferes with their power of contraction and dilatation, and influences the nutrition of the structures which they supply. This is affected, too, by a partial blocking of the arteries, also attributable to the swelling. The parenchymatous cells may atrophy as the result of pressure similarly produced.

The substance which is formed in the degenerate connective tissue, *lardacein*, is albuminous in nature, and not starchy, as the term "amyloid" would imply. It is probably a gluco-protein, the albumin moiety being in close combination with chondroitin-sulphuric acid. It is recognized by its staining properties, being stained mahogany brown by iodine, and rose-red or pinkish violet by methyl violet. The structures chiefly concerned are the kidney, spleen, liver, mucous membrane of the intestine, and the lymphatic glands. An affected organ is generally enlarged, pale, and hard, having somewhat the consistence of india-rubber. On section, the cut surface has a translucent, waxy appearance. In the case of the liver the degeneration is usually patchy in its distribution. Two forms are found in the spleen. In the commoner, the Malpighian follicles are especially affected, and appear like grains of boiled sago studded throughout the organ. Hence this form is known as the "sago" spleen. In the rarer, the walls of the venous sinuses and the reticulum are the degenerated parts. The organ is more enlarged, and the degeneration more diffuse.

AMYOTONIA CONGENITA

There is much variety in the appearances of amyloid kidneys, due to the fact that amyloid degeneration in that organ is usually associated with one or other form of nephritis. The kidney may be little altered in size or appearance, may be large, pale, and mottled, or may be small, firm, and granular.

Symptomatology.—Amyloid disease is recognized clinically by the occurrence of enlargement of the liver, enlargement of the spleen, albuminuria, and diarrhoea in a patient who is the subject of chronic suppuration, chronic tuberculosis, or advanced syphilis. Anæmia and wasting are usually present. The liver may reach to the level of the umbilicus or below it, is smooth, hard, and free from pain or tenderness. Its enlargement is accompanied neither by ascites nor by jaundice. The size of the spleen is greater when the degeneration is of the diffuse form. In such cases its enlargement may be considerable. Like the liver, it is smooth, firm, and painless. In the early stages of amyloid disease of the kidney, albuminuria may be absent. Later, albumin appears in the urine, increases in amount, and may become abundant. A considerable excess of urine is passed, perhaps well over 100 oz. in the twenty-four hours. It is clear, of low specific gravity, and contains various forms of the casts of degeneration—fatty, granular, and waxy. Œdema is often absent unless the anæmia and cachexia are profound. High arterial pressure, arterio-sclerosis, and hypertrophy of the left ventricle are not prominent signs, but occur in those cases in which chronic interstitial nephritis is an accompanying condition.

Treatment.—It is open to question whether amyloid degeneration ever disappears, but it probably does so in its earlier stages if the cause be removed. Alkalis are said to prevent its formation and hasten its absorption. Mercury and iodide of potassium are indicated in the syphilitic cases. The anæmia and general enfeeblement require tonic treatment, especially by iron. The diarrhoea is often very intractable. For this the usual remedies may be employed in vain. The most efficacious is probably opium in the form of pil. opii or pil. plumbi cum opio. **FREDERICK LANGMEAD.**

AMYOTONIA CONGENITA.—A disease of infancy characterized by extreme flaccidity of the muscles, which are small and weak. There is no true paralysis and no local wasting. It is most pronounced at birth, and tends slowly

and partially to improve, though recovery is unknown. The tendon reflexes are abolished completely. There are no other signs of involvement of the nervous system.

Etiology and morbid anatomy.—The cause is unknown. The disease is probably due to developmental defect of the muscles. Whether it is hereditary is not certain, but it may be present in more than one member of a family. Both sexes are affected, more commonly males. It is thought by some observers to belong to the myopathies, and is then classified as the simple atrophic type. The post-mortem findings in the two diseases are certainly very similar, and consist in defect and atrophy of muscles. Changes in the nervous system are rare.

Symptomatology.—The disease is congenital and is most severe at birth. The distal limb and small hand muscles are chiefly affected. Those of the face and of deglutition and phonation are intact. Very characteristic is the extreme tonelessness, smallness, and softness of the muscles. The flaccid state of these allows an extreme range of passive mobility at the joints, so that the limbs can be placed in abnormal positions, and the child, as it were, folded up. The patient is quite helpless for some time after it should begin to be able to hold its head upright and to sit up; and though some slow and partial gain of power is seen, the child is rarely, if ever, able to stand or walk alone.

Treatment.—The slight increase in strength can be reinforced by active and passive movement and exercise. A child who is unable to stand may develop considerable activity and mobility on a low cart on small wheels about 3 in. in diameter. Later, the ordinary child's walking-cart may be of use. During the early months of life massage is of value.

F. M. R. WALSHE.

AMYOTROPHIC LATERAL SCLEROSIS (see MUSCULAR ATROPHY. PROGRESSIVE).

ANÆMIA.—Under this title are included :

1. SECONDARY OR SYMPTOMATIC ANÆMIA.
2. CHLOROSIS.
3. PERNICIOUS ANÆMIA.
4. APLASTIC ANÆMIA.
5. CHRONIC SPLENIC ANÆMIA.
Gaucher's Disease.
Banti's Disease.
6. ANÆMIA PSEUDO-LEUKEMICA INFANTUM.

1. SECONDARY OR SYMPTOMATIC ANÆMIA

This heading covers those cases in which, as there is a definite cause for anæmia, the treatment can be based on the known etiological factor. Secondary anæmia may be (1) acute or (2) chronic.

(1) **Acute secondary anæmia** may be caused (a) by large hæmorrhages due to wounds, operations, gastro-intestinal ulcers, hepatic cirrhosis, splenic anæmia, ruptured ectopic gestation, hæmoptysis in pulmonary tuberculosis; or (b) by hæmolysis due to acute septicæmia and malaria, and to poisons such as potassium chlorate or nitrobenzol.

The **symptoms** are determined more by the rapidity with which the blood is lost than by the quantity; if moderate hæmorrhages are repeated there is time for the fluid part of the blood to be to some extent supplied, whereas a single large hæmorrhage, equal to or less in amount than that lost on several occasions, leaves the vascular system seriously depleted.

The symptoms are practically the same as those of secondary wound shock, or "ex-anæmia." The patient becomes pale, restless, yawns, feels faint, has tinnitus, blurring or loss of vision, tremors, nausea and vomiting, delirium, and perhaps convulsions. The pulse becomes rapid, weak and collapsing, and the blood-pressure very low.

Regeneration of the blood takes a variable time, and depends on the patient's previous health, the state of his tissues, especially that of the bone-marrow, and the store of iron. The fluid part of the blood is first made good, a restoration which may be correlated with the urgent thirst following hæmorrhage. Immediately after the hæmorrhage, examination of a drop of blood does not show any change; it is not until the fluids of the body have been attracted into the blood that the anæmia becomes obvious. The red blood-cells are then reduced in number, rarely below two millions, though patients with counts under a million have recovered. The red cells are irregular in shape and form, and deficient in hæmoglobin, so that the colour index is low; the activity of the bone-marrow is shown by polychromatophilia, normoblasts, and polymorphonuclear leucocytosis; and the platelets are increased in number. This is the usual condition, and is also seen in chronic secondary anæmia. After very severe hæmorrhage the hæmoglobin percentage may fall to 10, and a case that recovered had only 5 per cent. (Gulland and Goodall). In the most severe cases after repeated hæmor-

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rhages Cabot describes a blood picture resembling that of pernicious anæmia, except for the absence of megaloblasts.

Death may take place at once or after some days. Thrombosis may occur during convalescence, and death may be due to pulmonary embolism. The immediate prognosis depends on arresting the hæmorrhage and on efficient transfusion. The duration of convalescence naturally varies according to the patient's powers of recuperation.

Treatment.—Operation for arresting internal hæmorrhage has been thought inadvisable when the percentage of hæmoglobin has fallen below 30. Apart from stopping hæmorrhage, the most urgent necessity is to ensure an adequate supply of blood to the vital organs, especially the nerve-centres. This would most logically be effected by transfusion of blood, with due precautions both in the technique and as regards prevention of hæmolysis and agglutination of the red cells. Although there may be cases, especially those with very grave hæmorrhage, in which transfusion of blood or injection of preserved blood-corpuscles is more desirable, Bayliss has shown the value of intravenous injection of a 6-per-cent. solution of gum arabic in 0·9-per-cent. saline solution; a pint should be given as soon as possible, and another in half an hour if there be only partial improvement. The large reserve of hæmoglobin in the blood must be remembered in connexion with the success of gum-arabic solution as a substitute for blood, the important point being to maintain the circulation, which this solution does efficiently. Intravenous injection of isotonic and hypertonic salt solutions gives rise to temporary improvement only, for the fluid leaves the vessels in about half an hour. In the hæmorrhagic disease of the newly born (*melæna neonatorum*) subcutaneous or intramuscular injections of small quantities of blood or serum have given good results, thus suggesting that the bleeding is arrested or the bone-marrow stimulated thereby. Rest and warmth are obvious essentials for patients with post-hæmorrhagic anæmia.

Chronic secondary anæmia may be due to slight but long-continued loss of blood from gastro-intestinal ulcers, from ankylostomiasis, from piles and uterine hæmorrhages. Chronic suppuration and infections such as tuberculosis, syphilis, subacute bacterial endocarditis and malaria, new growths, and intoxications such as plumbism, mercurialism, and acetanilide

poisoning, and chronic renal and hepatic disease, are also familiar causes. Inanition, except that due to œsophageal or pyloric obstruction, is not a frequent cause in this country, but the war œdema among the Germans and their prisoners in the European War was accompanied by hydræmic anæmia, slight leucopenia, and relative lymphocytosis. Malignant disease may cause anæmia in several ways, by inanition, by repeated small hæmorrhages, or by providing autolytic products with hæmolytic properties.

Symptoms.—The patients are pale, wanting in physical and mental vigour, dyspeptic, constipated, easily tired and short of breath on exertion, subject to palpitation and faintness, and eventually show cutaneous hæmorrhages, signs of cardiac failure, and œdema of the legs. The blood changes have been described under Acute Secondary Anæmia. But some further points must be mentioned: in rare instances very grave anæmia resembling the pernicious type is seen in association with tapeworms and in malignant disease of the stomach. Usually the anæmia due to these conditions is ordinary secondary anæmia. In plumbism both the ordinary and the nucleated red blood-corpuscles show a characteristic stippling due to basophil granules even when the anæmia is not so profound as it may be in this condition.

The **treatment** should in the first place be directed to removal or, if this be impossible, mitigation of the cause. The general health should, of course, be carefully supervised.

2. CHLOROSIS

Chlorosis is an anæmia of unknown origin in young females, characterized by deficiency of hæmoglobin, a low colour index, and by rapid improvement when treated by iron.

Etiology.—Although its occurrence in males cannot be absolutely denied, it is so exceptional as to be practically negligible. Heredity plays some part in the incidence of the disease, and its occurrence in members of large families is compatible with the view that the blood change is an exaggeration of that most suitable for fertility (Lloyd-Jones). The onset coincides with puberty, and, though relapses may occur in the third decade, the onset is seldom, if ever, delayed beyond the second. Servants who have lately come from the country into towns are especially attacked. Of late years it has become curiously rare, at least in London, and is steadily diminishing in the United States, where the prevalence of patent medicines con-

taining iron has been suggested as the cause (Emerson).

The **pathogeny** is unknown. Virchow's view that it is due to hypoplasia of the heart and vascular system appears incompatible with its rapid cure by iron; and, except that chlorosis commonly appears about the time when menstruation begins, there is no reason to associate it with disorder of the ovaries or their internal secretion. Diet deficient in iron, bad hygienic conditions, and constipation have not been proved to be true causes. That hæmoly-sis does not play any part in its causation seems certain, and it is accordingly often stated that chlorosis is due to defective formation of the blood. D. Symmers suggests that the anatomical signs and the symptoms of chlorosis justify the conclusion that this form of anæmia is an incidental and fugitive condition in girls with status lymphaticus.

The blood.—On pricking the skin the blood wells out readily, is pale, and the coagulation time is prolonged. The specific gravity is diminished to 1030 from the normal of 1055 for girls, but that of the plasma is normal. Haldane and Lorrain Smith found that the volume of the blood is increased in proportion to the severity of the disease and that the increase is in the plasma, the total amount of hæmoglobin remaining unchanged. Ordinary examination of the blood shows a moderate diminution of the red cells, commonly to 4,000,000, with a much greater diminution in the amount of hæmoglobin to 35 and even to 20 per cent., so that the colour index is low—0.5 or less. The red cells are normal in shape but smaller than normal; poikilocytes and normoblasts are rare. The blood-platelets are much increased in number. The leucocyte count is normal in the absence of complications; some writers describe a relative increase in the lymphocytes. (Plate 2, Fig. 1.)

Clinical picture.—The onset is gradual, with shortness of breath and fatigue progressively increasing. The patients are well-nourished, brunettes having a yellowish pale skin and blondes a white complexion; occasionally, however, the cheeks remain red though the anæmia is considerable. The sclerotics are bluish, and there may be pigmentation about the joints, but there is no trace of jaundice. Oedema of the eyelids and feet is common. Headache, tinnitus, dizziness, neuralgia, and mental irritability are not infrequent, and amenorrhœa, dysmenorrhœa, some disturbance of menstruation or leucorrhœa is the rule.

Slight fever may be present. Not uncommonly chlorotic girls are drowsy when up and cannot sleep when in bed. Faint-feelings are common, and there is palpitation. The pulse is full, soft, and of normal or slightly low blood-pressure. A *bruit de diable* over the jugulars is best heard on the right side of the neck on inspiration. A systolic murmur is usually present over the cardiac area, louder when the patient is lying down, and as a rule most intense over the pulmonary artery; it is generally soft, but is occasionally loud and harsh; it may also be heard at the apex, or at both the apex and the base. The cardiac dullness is usually increased upwards, and there may be pulsation over the upper part of the right ventricle where the pulmonary artery arises. The origin of these hæmic murmurs has been much discussed. The urine is pale, poor in pigments, and often contains a trace of albumin. The appetite is capricious, and may be perverted, with a craving for acids, chalk, or earth (pica). Hyperchlorhydria with the accompanying pain is common, and it has usually been considered—though this has been disputed by C. Bolton—that chlorosis disposes to gastric ulcer. Dyspepsia, with atonic dilatation and downward displacement of the stomach, is a frequent and early event. Constipation, which is the result and not the cause of the chlorosis, is present in the great majority of the cases, and not infrequently induces piles.

Complications.—Venous thrombosis, probably infective in origin, occurs in about 2 per cent. of all cases, usually in the legs, and may then cause pulmonary embolism. Thrombosis occasionally involves the cerebral sinuses and veins, causing intense headache, convulsions, optic neuritis, hemiplegia, coma, and death, but at first the symptoms may suggest hysteria. In less severe cases recovery, with or without hemiplegia, may follow. Optic neuritis in chlorosis may be due to otherwise latent thrombosis of the cerebral sinuses. Signs of exophthalmic goitre, usually in an incomplete form, complicate a small proportion of the cases.

Diagnosis must be made from the secondary anæmias which show a similar chlorotic type of the blood, but with leucocytosis and a colour index that is not so low. It is particularly important to distinguish chlorosis from the secondary anæmia associated with incipient pulmonary tuberculosis. As signs of hyperthyroidism are not uncommon in chlorosis, the distinction from early exophthalmic goitre must be remembered. The oedema may suggest renal

PLATE 2.—THE BLOOD IN (1) CHLOROSIS,
(2) PERNICIOUS ANÆMIA. × 500.

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disease. If a case does not rapidly improve on efficient treatment the diagnosis should be reconsidered and search made for some definite cause for secondary anæmia.

Prognosis.—The disease is readily curable by iron, but is extremely prone to relapse, especially if the treatment be prematurely abandoned. It rarely persists after marriage, and, except for accidents due to thrombosis, is never fatal.

The **treatment** is by iron, preferably in the form of Bland's 5-gr. pill of ferrous sulphate and carbonate of potassium, taken three times daily after meals, one during the first, two during the second, and three during the third week and for four or five successive weeks before the amount is diminished. The treatment should be persisted in for three or four months at least, although the patient may be so well that difficulties arise. Constipation must be prevented, especially during the administration of iron. Occasionally the stomach does not tolerate this preparation, and freshly prepared reduced iron (trochisci ferri redacti of the British Pharmacopœia, 1 gr.) or the scale preparations (e.g. ferri et ammonii citratis 7½ gr. with a drachm of syrup in a tablespoonful of water, three times daily after food) must be substituted for a time. Organic preparations of iron and hæmoglobin are unsatisfactory, and hypodermic injections of iron are rarely found necessary. Purgation has been tried on account of Haldane and Lorrain Smith's determination of the excessive amount of plasma, but without success. In severe cases rest in bed for three weeks facilitates the cure.

3. PERNICIOUS ANÆMIA (*syn.* ADDISONIAN ANÆMIA ; PROGRESSIVE PERNICIOUS ANÆMIA)

A severe, relapsing, and ultimately fatal anæmia of hæmolytic but otherwise obscure nature, with characteristic blood changes.

Etiology and pathology.—Pernicious anæmia is a disease of middle life, three-quarters of the cases occurring after the age of 36, and is twice as common in males as in females. Pregnancy has been thought to be a factor, but some of the reputed cases may really be examples of grave secondary anæmia.

The hæmolytic nature of pernicious anæmia is generally accepted; Hunter localized the hæmolysis in the portal area, on the grounds that the remains of the liberated hæmoglobin were specially obvious in the liver in the form

of hæmosiderin, and that destruction of blood in the general circulation would be likely to be shown by hæmoglobinuria, but this conclusion has been much debated. Hæmolytic agents introduced into the general circulation produce deposits of hæmosiderin in the liver and spleen just as in pernicious anæmia, because this transformation takes place there, but it is probable that the hæmolytic and phagocytic powers of the spleen, lymphatic glands, and bone-marrow are pathologically exaggerated by the poison and that the actual hæmolysis occurs in the hæmopoietic centres; further, it has been shown that the kidneys only excrete hæmoglobin when its content in the serum reaches a certain level.

The hæmolysis may be due to various causes. Thus, streptococcic infection of the stomach from pyorrhœa (Hunter), subinfection with *Bacillus coli* (Adami), excessive activity of *Bacillus welchii* in the colon (Herter), and protozoan infection have each been invoked. Dibothriocephalus anæmia, which so closely resembles pernicious anæmia, is thought to be due to a hæmolytic lipid. It might, therefore, be considered that pernicious anæmia is not a disease due to a specific poison, but a symptom complex which may be brought about in several ways.

Morbid anatomy.—The fat, which is considerable in amount, is bright yellow in colour; the tissues are œdematous, and the serous cavities contain fluid. All the organs are pale and anæmic, but the muscles may be dark red. The heart is well covered with epicardial fat, and the myocardium, especially the musculi papillares, shows fatty degeneration ("tabby-cat striation"). The marrow of the long bones is hyperplastic, bright red in colour, and soft. Microscopically it shows an excess of megakaryoblasts, thus contrasting with the normoblastic reaction of the red bone-marrow in secondary anæmia. The liver is fatty, and Perl's test shows a deposit of hæmosiderin in the cells in the periphery of the lobules. The spleen varies in size; usually it is slightly enlarged, but as the disease progresses the spleen gets smaller. There is usually excess of hæmosiderin, the iron content being three times that of the normal. The hæmolymp glands may be prominent in size and number, but this is not constant. The kidneys may contain hæmosiderin, especially in the cells of the convoluted tubules; and it is in these cases that casts containing hæmosiderin are found in the urine (P. Rous). In some instances the gastric mucous membrane shows

atrophy, but this is more probably a result than the cause of the disease.

The brain shows focal degeneration in the white and grey matter, beginning around the pyramidal cells of the marginal grey matter, and as a result of destruction of these cells the medullated fibres in the white matter undergo secondary degeneration. The plaques of degeneration are regarded by Woltmann as toxic in origin. Small foci of degeneration in the spinal cord appear first in the posterior, then in the lateral columns, and eventually become confluent; the change is most prominent in the cervical region, and is commonly present after death, even in cases in which there were no corresponding symptoms noticed during life. These lesions were found in 82 of Cabot's 96 cases, or 85 per cent., and in 77 per cent. of Minnich's cases. As these cord changes—subacute combined degeneration—may occur before or without any anæmia, James Collier argues that they are not due to the anæmia, but that both the anæmia and the nervous degeneration are related not as cause and effect but as concomitant results of a pathological process which may be responsible for more than one toxin.

Blood.—The specific gravity is low (1030 instead of 1056), the amount of water and NaCl being increased, and of solids, potassium and iron decreased. The total amount of the blood is much diminished, and there may be difficulty in obtaining it by puncture. The drop of blood is abnormally fluid, but may be of a good colour from the high colour-index. The number of the red blood-corpuscles is much reduced—to below 2,000,000, and even down to 500,000. The cells vary in size (those much increased in size—megalocytes—being almost pathognomonic), show poikilocytosis, diffuse polychromatophilia and stippling. The resistance of the red cells to hæmolytic salt solutions is increased. Nucleated red cells—normoblasts and megaloblasts—are constant, the latter being specially important. The number of erythroblasts varies, and large numbers (blood crises) may appear in the circulation for a short time. The hæmoglobin is much reduced and may fall to 10 per cent., the reduction being proportional to the severity of the disease. The colour-index is characteristically high, being over 1 and approaching 2. This is due to the increased amount of hæmoglobin in the red cells, particularly the megalocytes. The blood-counts and hæmoglobin percentages run parallel.

With the advance of the disease the colour-index mounts higher, whereas in remissions it falls. The leucocytes are diminished, especially the polymorphonuclears, so that the lymphocytes appear to be relatively increased. Occasionally there is slight eosinophilia. There is an unimportant increase in the number of myelocytes. The platelets are much diminished. The essential diagnostic points are the great diminution of the red blood-corpuscles, the high colour-index, the presence of megaloblasts and megalocytes, and leucopenia. (Plate 2, Fig. 2.)

Symptomatology. A special feature of the disease is the occurrence of remissions and exacerbations, so that though on the whole the term progressive pernicious anæmia is justified, the course is not one of uniform deterioration. There may be as many as six remissions during which the hæmolytic process is arrested and the symptoms disappear, the blood sometimes becoming normal though often showing some evidence, such as large red cells, of the latent disorder. The first and the early remissions are more complete than the later ones. The remissions may come on suddenly. The onset is, as a rule, gradual, with increasing pallor, weakness, and breathlessness on exertion, but in rare instances the symptoms appear to date from an acute gastro-intestinal attack. The waxy pallor of the skin is combined with a lemon-yellow tint. Occasionally the skin shows brownish pigmentation. There is œdema of the feet, and occasionally general anasarca appears rapidly. Small hæmorrhages may be seen in the skin and in the retina, but are not so common as in aplastic anæmia. I have seen conjunctival without retinal hæmorrhages. The mucous membranes seem bloodless, and in rare instances the oral mucosa may show pigmentation before arsenic has been taken. The tongue may be sore, with superficial glossitis and ulceration, especially during the periodic exacerbations. Oral sepsis is by no means constant. The appetite fails, attacks of abdominal pain may occur, and vomiting and diarrhœa, not necessarily due to arsenic, though this contingency should always be borne in mind, may be troublesome; diarrhœa is said to occur in half the cases, and sometimes to be followed by improvement. The urobilin in the fæces is increased during active hæmolytic, and is an index of its degree. Considerable gastro-intestinal hæmorrhages are rare. The hydrochloric acid in the gastric juice is deficient or absent. The spleen is palpably enlarged in a third and the

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liver in a third to a fourth of the cases. Definite jaundice is rare, and almost always due to some complication. The pulse is somewhat collapsing and of low blood-pressure; there may be excessive arterial throbbing, and palpitation is common. The heart may be dilated, and systolic murmurs are almost the rule. Muscular weakness is a striking and often an early symptom, but occasionally a patient with very grave anæmia is able to continue at work. The subcutaneous fat is well maintained, so that the patients, though flabby, are very well nourished, and the contrast between their apparent nutrition and real weakness and anæmia is remarkable. The bones may be rather tender. Low, irregular fever may persist for weeks when the disease is advancing, and the temperature may be higher during the exacerbations. Tingling in the extremities is estimated by Woltmann to occur in 80 per cent. of the cases; its relation to arsenical neuritis should be remembered. Deep sensation, especially of vibration and joint sensibility, is impaired, cramps may be troublesome, and more definite evidence of the changes commonly present in the spinal cord may be shown by the presence of subacute combined degeneration with spastic or tabetic symptoms. Mental dullness and apathy are common in advanced stages and may be correlated with the degenerative foci in the brain. Definite psychoses are rare, and are probably toxic in origin.

The urine is usually normal in amount and pale; though Hunter and others have described a dark colour due to pathological excess of urobilin, especially during the exacerbations. Albumin in small quantities is not uncommon, but casts are somewhat rare. The iron is much increased, and hæmosiderin may be found in renal casts and cells (P. Rous).

Death from increasing weakness is often preceded by semicoma for some days. A fatal termination may be precipitated by pneumonia sometimes almost latent.

Diagnosis depends on an accurate blood examination, and in cases seen during a remission several blood examinations may be necessary. Some cases of *latent carcinoma of the stomach* without a palpable tumour may have a lemon-tinted skin and clinical symptoms which strongly suggest pernicious anæmia, but such cases rarely have a red blood-count under 1,500,000, or a high colour-index, or megaloblasts. In grave *postmalarial anæmia* the blood picture may be that of pernicious anæmia. In some cases of *acquired hæmolytic*

jaundice the blood may resemble that of pernicious anæmia and a distinction may be difficult, though the corpuscular resistance should be low in hæmolytic jaundice.

Prognosis is practically fatal; Cabot regards 3 out of 1,200 cases in which no relapse occurred for six years as cures. As a rule, life is not prolonged for more than three years after the onset, and many cases prove fatal in a much shorter time. The acute, subacute, or chronic nature of the case naturally influences the opinion as to the duration of life, and this also depends on the duration of the remissions. The number and duration of the remissions vary considerably; out of 329 cases in which the remissions were accurately recorded, Cabot found that a year was the most frequent duration of a remission; the first remission is most likely to be complete also. The cases associated with *Dibothriocephalus latus* are said to be curable if the worm be removed.

Treatment.—Brilliant results have been recorded after almost every form of treatment, but it is difficult to eliminate the possibility that a spontaneous remission coincided with the treatment. It is disappointing to find that an unbiased investigation by Bloomfield at the Johns Hopkins Hospital did not show any evidence that arsenic, transfusion of blood, splenectomy, or elimination of infective foci prolongs life in this disease. Cabot also doubts if any drug, even arsenic, has any considerable influence over its course. Arsenic in the form of Fowler's solution, beginning with 2 min. and working up to 15 min. three times a day after food, is usually employed. A watch must be kept for its toxic effects on the alimentary canal, such as vomiting and diarrhoea, and for peripheral neuritis. Intramuscular injections of salvarsan preparations have been recommended by Byrom Bramwell, who introduced the use of arsenic in 1875, and reports laudatory and deprecatory have been published. Cacodylate of sodium, bone-marrow, and inhalations of oxygen are placebos with less to recommend them. More recently splenectomy has been practised but, though vaunted at first, is now in the stage of considerable criticism, and has been abandoned at the Johns Hopkins Hospital. Among W. J. Mayo's 50 cases of splenectomy the operative mortality was 3, or 6 per cent., but in 10, or 20 per cent., splenectomy prolonged life. Transfusion at the Johns Hopkins Hospital in cases already splenectomized did not show that the blood was

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"held" better than in other cases. Transfusion of blood has been extensively employed, and has been described by Ottenberg and Libman as the best remedy but not a cure for pernicious anæmia, as it causes remissions in half the cases. Transfusion of moderate amounts, 500 to 1,000 c.c., should be made after a Wassermann test has been performed on the donor and tests to eliminate hæmolysis and agglutination have been carried out. Hæmolysis is likely to occur in repeated transfusion. At one time antistreptococcic serum and simple horse-serum were employed. Small doses of benzol and of X-rays have been recommended on account of their stimulating influence on the bone-marrow, but their effects must be carefully watched. Glycerin, intestinal antiseptics such as salol and β -naphthol, vaccines, and free purgation have also been recommended. Iron probably does more harm than good.

Rest in bed, with as much fresh air and sunlight as possible, is advisable. Feeding may be difficult from the distaste for food, but it does not appear that restriction of proteins in order to minimize intestinal putrefaction is really effective. In spite of a philosophic doubt as to its efficacy, arsenic should be cautiously pressed.

4. APLASTIC ANÆMIA

In this grave form or phase of anæmia the reaction of the bone-marrow is defective.

Pathogenesis.—Aplastic anæmia has usually been regarded as a form of pernicious anæmia in which the red bone-marrow fails to regenerate the blood. But recently J. H. Musser, jun., and others argue that there is no evidence of hæmolysis, such as would be provided by jaundice, urobilinuria, or visceral hæmosiderosis, and that aplastic anæmia is entirely due to aplasia of the bone-marrow which is caused by the selective action of a poison (myelotoxin). In other words, that aplastic anæmia is distinct from pernicious anæmia. It is known that benzol and trinitrotoluene cause aplastic anæmia; benzol poisoning is characterized by purpura hæmorrhagica, almost complete absence of blood-platelets, and progressive leucopenia, which persists after the cause is removed. In the case of trinitrotoluene, B. Moore inclines to the view that aplastic anæmia results from exhaustion of the bone-marrow due to increased strain entailed by the increased hæmoglobin metabolism. Infections such as stomatitis may produce a poison which, either from its virulence or because the

resistance of the bone-marrow is inherently or temporarily impaired, paralyses or destroys the hæmatopoietic tissues.

Morbid anatomy.—The red bone-marrow is yellowish-white, fatty, and contains few if any cells. Hæmosiderosis, or deposit of iron in the liver and other viscera, which is such a noteworthy feature of pernicious anæmia, has rarely been noted. The spleen resembles that in pernicious anæmia. The serous membranes show numerous punctate hæmorrhages.

Symptomatology.—Grave anæmia without any evidence of regeneration of the blood is the characteristic feature of the disease, which resembles pernicious anæmia except that its course is acute and without remissions. The number of red cells is reduced to a million or less; the colour-index is usually under—often considerably under—one; signs of activity of the bone-marrow—such as nucleated red cells, myelocytes, myeloblasts, polychromatophilia, and anisocytosis—are absent; and the platelets are diminished to 25 per cent. or less of the normal. There is leucopenia to 3,000 or much below, and the granular cells are absolutely reduced (polymorphonuclears being 8–20 per cent.), so that there is a relative lymphocytosis. The disease is essentially one of early adult life; among L. W. Smith's 61 collected cases the average age was 29½ years, and the sexes were equally attacked. It is specially prone to be accompanied by cutaneous and mucous hæmorrhages, which may be so extensive as to suggest that they are the cause of the anæmia, and by fever, which is often higher than in other forms of anæmia. The lemon tint of pernicious anæmia is not seen in the skin or conjunctivæ. There is no hepatic or splenic enlargement. The disease runs a rapid course in a few weeks or months; the average duration of Smith's 50 collected cases was three months, and out of Cabot's 24 cases 5 only lasted longer than this.

A chronic aplastic anæmia (hypoplastikæmia) has been described as due to exhaustion of the bone-marrow, either inherited or acquired, with the result that the bone-marrow is unable to maintain the normal standard of the blood (Parkes Weber).

Diagnosis.—It has generally been considered that a certain diagnosis can be made only by inspection of the bone-marrow, but Musser, who has analysed 59 cases, considers that the clinical and hæmatological features are sufficient. A careful blood examination may be necessary to distinguish the disease from acute

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lymphoid leukæmia, especially in an aleukæmic phase when the total white count is not raised, for casual examination of a stained film may suggest the latter disease. In young children the condition must also be distinguished from von Jaksch's anæmia by examination of the blood.

The **prognosis** is almost uniformly hopeless, but, assuming that the life of the red blood-corpuscle is between Zoga's estimate of ten and Ashby's of thirty days, the whole of the bone-marrow is not destroyed or paralysed at once, at least in those cases lasting for more than a month. It is therefore important to remove any continuous source of poison such as benzol, trinitrotoluene, or toxins from an infective focus.

Treatment in the past has been on the same lines as for pernicious anæmia, but it would appear to be advisable to avoid the use of arsenic, which would stimulate the exhausted bone-marrow, and to rely on transfusion of blood from a donor of the same group or of Group IV. The patient should be kept absolutely at rest.

5. CHRONIC SPLENIC ANÆMIA

A condition characterized by (1) chronic splenomegaly which cannot be correlated with any recognized cause; (2) absence of enlargement of lymphatic glands; (3) chlorotic anæmia; (4) absence of leucocytosis and usually the presence of leucopenia; (5) periodic gastro-intestinal hæmorrhages; (6) prolonged course without any tendency to spontaneous cure, though splenectomy—if successful—is usually curative.

Etiology.—It is not hereditary or familial. In this country females about puberty are rather more often attacked than others, but the sex- and age-incidence vary considerably. The cause is unknown; a streptothrix and a pleomorphic diphtheroid organism (*Bacillus hodgkini*), also found in lymphadenoma by Bunting and Yates, have been described, but confirmation is required. At present it may be assumed that a chronic toxic or infective process is located in the spleen, which therefore undergoes fibrotic enlargement, and that the large spleen mechanically causes gastric hæmorrhage and so anæmia.

Morbid anatomy.—The splenic enlargement is considerable; the organ, on an average, weighs under 2 lb., and so is very much less than in Gaucher's disease (see p. 62). It may show adhesions and pericapsulitis. The splenic

vein is enlarged, tortuous, and often shows endophlebitis. The vasa brevia in the gastro-splenic omentum are dilated, and the veins at the lower end of the œsophagus become varicose and sometimes ulcerated. Microscopically the spleen shows chronic diffuse inflammation both of the connective and parenchymatous tissues.

Symptomatology.—The onset is gradual and the course very chronic. The cardinal features are splenomegaly, hæmorrhages, and the blood changes. The splenic enlargement always precedes the anæmia, sometimes by a very long interval. Periodic gastro-intestinal hæmorrhages are probably brought about mechanically by kinking of the vasa brevia in the gastro-hepatic omentum, the blood often coming from an œsophageal varix. The amount lost may be very large, or prove fatal, and the resulting anæmia be very profound. The anæmia is of the chlorotic type, with a low colour-index. The leucocytes are not increased in number, and, indeed, are usually diminished. In some cases there is a relative lymphocytosis. There may be a few myelocytes in the blood. The lymphatic glands are not enlarged, and the liver is not increased in size unless cirrhosis (Banti's disease) has supervened. The skin is sometimes pigmented, either diffusely or only on the abdomen. Œdema of the feet, cardiac murmurs, slight jaundice, and ascites without hepatic cirrhosis are sometimes described. Indigestion and abdominal discomfort may be present.

Diagnosis must be made from other forms of splenomegaly with anæmia. *Leukæmia* can be recognized by a blood-count. In latent *portal cirrhosis* the spleen is seldom so large as in splenic anæmia. The metasplenomegalic form of *biliary cirrhosis*, in which splenic precedes hepatic enlargement, is characterized by persistent jaundice. Chronic *obstruction of the splenic vein* exactly imitates splenic anæmia. *Syphilis of the liver and spleen* can be recognized by the Wassermann reaction. *Lymphadenoma* nearly always gives rise to enlargement of lymphatic glands, and so can be recognized. Chronic *infective endocarditis* sometimes simulates splenic anæmia very closely, but in such cases persistent albuminuria or hæmaturia may be present.

Massive tuberculosis of the spleen, in which there is very considerable enlargement without much evidence of tuberculosis elsewhere, is rare, but may imitate splenic anæmia, from which it differs mainly in the greater premi-

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nence of splenic pain and in the presence of hepatic and glandular enlargement. Chronic *splenomegalic hæmolytic jaundice*, which may closely resemble it in the periods when jaundice is slight, can be recognized by the determination of the fragility of the red blood-corpuscles.

Prognosis.—The disease may last for ten years, but usually is not so prolonged; as a rule when hæmorrhage has occurred the outlook becomes serious, as death may follow. Ascites usually means that hepatic cirrhosis (Banti's disease) has supervened. If splenectomy is successfully performed the outlook is good, otherwise there is little chance of cure.

Treatment.—Medical treatment consists in good hygienic conditions, iron and arsenic by the mouth, and salvarsan intravenously. The application of X-rays to the splenic region has been followed by diminution in the size of the organ, but it has not been proved that the benefit is permanent. Transfusion of blood may be useful in enabling a very anæmic patient to undergo splenectomy, but should not be regarded as a curative measure. Splenectomy is the only really successful form of treatment; but widespread adhesions make it difficult, and there is a risk of severe or fatal postoperative hæmorrhage. The operative mortality has been estimated at 12·5 per cent. In another series of 27 cases—at the Mayo clinic—there were 3 operative deaths and a total of 8 deaths in ten years, hæmorrhage being fatal in 2 cases, one five and a half years after operation; in 3 other cases hæmorrhage occurred two or three times after operation, but the patients eventually got well. Out of 61 cases operated upon, death occurred in 7, or 11·7 per cent. (W. J. Mayo).

GAUCHER'S DISEASE, OR LARGE-CELLED SPLENOMEGALY

This rare condition has generally been included as a subgroup of chronic splenic anæmia, from which, however, it differs widely both in its morbid anatomy and its clinical characters. Its special features are its familial but not hereditary occurrence in childhood, the large size of the spleen (averaging 7 lb.) followed by similar hepatomegaly; the presence in the spleen, liver, bone-marrow, and lymphatic glands of large cells measuring 20–40 μ or even more; its very chronic course, averaging nineteen years; a brownish-yellow discoloration of the skin; a yellowish wedge-shaped thickening of the conjunctivæ; the late incidence of hæmorrhages and anæmia; and pro-

gressive leucopenia from the start. Jaundice does not occur, and ascites is exceptional. Out of 19 authentic cases, 6 only were males. For the certain diagnosis of Gaucher's disease, detection by the microscope of the characteristic cells is necessary. As Mandlebaum points out, these cells must be distinguished from the large cells derived from the reticular cells of the hæmopoietic organs, as the result of various agents, bacterial, toxic, metabolic, or chemical. Thus, in diabetic lipoidæmia, cells in the spleen, and, in animals fed on fat or cholesterol, cells showing some resemblance to the Gaucher cells have been found. Gaucher cells, however, show structural differences, and never contain fats or lipoids. Splenectomy gives the best results, but, as the morbid process is not confined to that organ, cannot be considered as a radical cure.

BANTI'S DISEASE

Though often used as a synonym for splenic anæmia, this name should be restricted to those cases in which hepatic cirrhosis and ascites have supervened. Banti described three stages of "splenomegaly with cirrhosis": the first, or preascitic, lasting three to five years, is splenic anæmia; in the second or transitional stage, lasting a year to a year and a half, anæmia, ill-health, gastro-intestinal disturbance, diarrhœa, and piles appear; the third stage, which may rightly be called Banti's disease, is characterized by cirrhosis and contraction of the liver, ascites, hæmorrhages, and wasting. The morbid anatomy is that of splenic anæmia with multilobular cirrhosis of the liver in addition. Diagnosis must be made from ordinary multilobular cirrhosis by the history, and from syphilis of the liver and spleen by the Wassermann reaction. Treatment is on the same lines as in splenic anæmia, but splenectomy, which gives the only chance of recovery, is attended by a much higher mortality, probably about 50 per cent.

6. ANÆMIA PSEUDO-LEUKÆMICA INFANTUM (VON JAKSCH)

Splenomegaly with grave anæmia and myelæmia in infants, from which recovery usually occurs. It is sometimes called Splenic Anæmia of Infants.

Historical note.—The condition was described by von Jaksch in 1889, but its existence as a definite disease rather than as a form of secondary anæmia due to various causes has been much debated. At present the general

view appears to be that, though it may not be a definite disease, it is convenient to recognize it as a clinical entity.

Etiology.—It occurs within the first three years of life, usually beginning about the end of the first year, and attacks males more often than females. It is not hereditary, but twins have been noted to be affected, probably from the similarity of their circumstances. It is a disease of the poor, and has been thought to be favoured by prolonged suckling and to be specially frequent in Jews. It may be associated with evidence of rickets, or chronic gastro-intestinal disturbance. In spite of its resemblance to infantile kala-azar, there is no evidence that it is due to any definite infection, and it has been thought that it is not a special disease but only a collection of symptoms or a syndrome which may be produced by various factors, and that its differences from chronic secondary anæmia are due to the manner in which the bone-marrow reacts in infancy.

Morbid anatomy.—The spleen, which may weigh eight or ten times more than normal, is firm, and on section and microscopically may show fibrosis. The lymphatic glands may show a similar change, and the liver some increase in the contained fat. In some cases regarded during life as infantile splenic anæmia, myeloid transformation in the spleen and liver has been found; thus suggesting that the cases were really examples of leukaemia. The bone-marrow shows some hyperplasia.

Symptomatology.—The onset is gradual and the course of the disease chronic. The skin presents a transparent pallor with a greenish yellow hue, and the mucous membranes are blanched. The spleen, which commonly reaches down to the crest of the ilium, usually is very considerably enlarged when the infant comes under observation, and is mainly responsible for the protuberant abdomen. The liver and lymphatic glands seldom show more than slight enlargement. Epistaxis, which may be the earliest symptom, and small cutaneous hæmorrhages are common. There may be puffiness of the face and extremities, which varies from time to time, and the urine may show a trace of albumin. Irregular fever, rarely about 102° F., may occur. Ascites and jaundice are not seen. There may be evidence of rickets; diarrhoea and broncho-pneumonia are frequent complications, and are the usual causes of a fatal termination.

The blood.—The erythrocytes are diminished to between two and three million per c.mm.

and the colour-index is low. Nucleated red blood-corpuscles—normoblasts in greater number than megaloblasts—are common. Poikilocytes, megalocytes and microcytes, and basophil and polychromatophil degeneration occur. There is usually leucocytosis, which may reach 50,000 but is commonly under 30,000. The mononuclear cells are generally increased, and myelocytes—about 5 per cent.—are almost constantly found. Thursfield refers to cases in which a terminal lymphocytosis produced the blood picture of acute lymphoid leukaemia.

Diagnosis.—It is important to recognize cases of secondary anæmia due to congenital syphilis, tuberculosis, rickets, and other causes, for in the presence of such a factor a case of supposed infantile splenic anæmia should be transferred to that category and treated on the appropriate lines. The spleen is seldom so large in these conditions, and the Wassermann and tuberculin tests will be valuable guides. Ordinary anæmia and myeloid leukaemia can usually be eliminated by a blood-count, but there appear to be transitional cases between the two diseases.

Prognosis.—The course of the disease is very chronic, but with suitable treatment recovery usually occurs, according to Thursfield, in 65–70 per cent. of all the cases. The anæmia disappears before the splenomegaly; in some cases both persist into adolescence. The onset of complications, such as broncho-pneumonia, is ominous.

Treatment.—Good hygienic conditions such as fresh air and sun, and careful dieting, with plenty of protein such as raw-meat juice, scraped meat, and eggs, are more effective than medicinal treatment. Hutchison speaks highly of oat flour. Cod-liver oil and iron should be employed, but arsenic is disappointing. For diarrhoea intestinal antiseptics may be tried. Splenectomy appears to be unnecessary in a disease in which recovery so frequently follows less heroic treatment. H. D. ROLLESTON.

ANÆSTHETICS. Selection of the anæsthetic.—In order to ensure successful results from the administration of anæsthetics, the first necessity is to choose the most suitable agent and give it in the most appropriate way in every particular case. The chief considerations are (1) the condition of the patient and his general physique, and (2) the site and nature of the operation.

1. **Condition of the patient.**—For a patient in normal health and having an operation of an

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special severity, ether is the best anæsthetic on which to rely. For the greater comfort and convenience of the patient it may be preceded by nitrous oxide, or ethyl chloride, or various alkaloids. The physique and the temperament of the patient must be considered in their bearing upon the way in which the anæsthetic shall be administered. Ether is given according to two main methods—(1) the open, (2) the closed. In such an operation as the radical cure of hernia the closed method should be chosen if it suits the patient's physique, because (1) it admits of a quicker and a more tasteless induction, (2) a full degree of narcosis is more rapidly attained, (3) after-effects are slight in such a case as we are considering, (4) the objections of cyanosis, free mucous secretion, etc., are avoidable, in an operation of no great length, with the preliminary use of atropine. If the patient is obese, short-necked, and florid, or if he has marked inability to breathe through his nose, closed methods are best avoided. Similarly, if he is highly nervous and apprehensive, success is achieved more conveniently by a gradual rather than by a sudden method of induction. For a normal adult, then, undergoing an operation of about half an hour's duration, gas and ether may be chosen as the routine method. For children under about 6 I prefer the open method, initiated by small quantities of C.E. mixture, and for old persons I generally select the latter mixture throughout. With young children it is usually desirable to give the anæsthetic while the child lies in bed and to carry him, when unconscious, into the operating room. The less he knows about any unusual arrangements the better.

Now let us consider the position of the person whose condition is not that of normal health. In the case of the *head and neck* our procedure may be modified by partial unconsciousness or delirium resulting from accidents; here chloroform gradually given is best, and the anæsthetic must be used very sparingly. It may also be modified by difficulty in breathing due to mechanical narrowing of the airway, as in tumours of the neck, diphtheria, laryngeal growths, etc.; here chloroform in diluted form is always to be chosen. Some ether may be used upon the open mask from time to time, but all closed methods must be rigorously avoided. The mouth should be opened slightly by a gag before the administration begins. *Disease within the chest*, if it affects the lungs, generally makes the use of closed ether un-

desirable, if not impossible. In chronic cases C.E. and, often, open ether in addition answer well. In acute conditions chloroform given with oxygen is generally the anæsthetic of choice. In active phthisis ether should be avoided altogether. In *cardiac affections*, where compensation is complete, the anæsthetic can be chosen on the usual grounds. Where there are failing compensation and dyspnoea all anæsthetics have added danger; this is diminished by the use of open ether and oxygen. Usually, the administration should begin with C.E. mixture. *Abdominal distension* also prevents the employment of closed methods if it is so marked as to interfere with the movements of the diaphragm; C.E. and open ether are best for such cases. It is well to keep the narcosis light till the distension has been reduced, as by letting out fluid, delivery of a cyst, or opening of a distended gut. In *diabetes* the sugar should be reduced by dieting, etc., as far as possible, before operation. Local analgesia should be chosen when applicable. Amputations have been successfully performed under gas and oxygen with the help of local analgesics, and the method seems admirable. The danger of general anæsthesia is that coma is likely to follow.

2. *Site and nature of the operation.*—Operations upon the *brain* are best performed either under chloroform, using Vernon Harcourt's method or a Junker, or under infusion anæsthesia. Narcosis is deep only for making the flap and for incising the dura mater. Often the anæsthetic can be discontinued till sewing up begins. *Eye* operations requiring general anæsthesia may be performed under ether when additional vascularity is unimportant. Thus, excisions of the globe and operations for squint are conveniently performed when a full ether anæsthesia has first been instituted by gas and ether or by other means. Chloroform is better for iridectomies and similar procedures, a special mask being used which just fits over the nose and mouth and permits continuous drop administration while the operator is at work. Operations upon the *tongue, fauces, jaws*, etc., unless of quite short duration, require special arrangements so that the anæsthetic may be continued during the operation. The older method, which is satisfactory, consists of first obtaining a full ether anæsthesia and then continuing with chloroform, delivered through a tube from a Junker's inhaler. Crile's tubes introduced into the naso-pharynx have made continuous administration of ether pos-

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sible in such cases. The method is excellent in feeble subjects. Again, infusion anæsthesia is especially applicable to these cases. Better still, in expert hands, is the use of intratracheal insufflation of ether. For removal of *tonsils and adenoids*, gas and oxygen or ethyl chloride are most suitable, if the guillotine only is to be used and the operator and anæsthetist are accustomed to working together. For enucleations or with a strange anæsthetist, C.E. mixture is far more certain to give a sufficient anæsthesia, which may be continued with chloroform from Junker's inhaler tube. Better still is a very deep ether narcosis secured by the open method. For operations upon the *larynx* chloroform is given from a Junker's tube inserted into the upper opening of the tracheotomy tube, for preliminary tracheotomy is necessary in most of these cases. Infusion anæsthesia can be employed if preferred. For *bronchoscopy* and similar procedures chloroform is the anæsthetic of choice, and after anæsthesia has been reached it is maintained by the delivery of the vapour down the bronchoscope from a Junker's tube. It is important to obtain deep narcosis in the first instance before any attempt is made to pass the bronchoscope into the larynx. Operations upon the *neck, including goitre*, may be most serious from the anæsthetist's point of view. Usually closed methods are to be avoided and reliance placed upon open ether preceded by morphia and atropine. Those accustomed to its use prefer intratracheal insufflation. Crile's method for using gas and oxygen, after the patient has practised taking it for several days, in conjunction with local analgesics and hypodermic injections, is especially recommended for the worst forms of exophthalmic goitre. Such cases are dangerous, no matter what form of general anæsthesia is used. Operations upon the *chest*, if associated with a free removal of chest-wall and opening of the pleura, should be done under intratracheal insufflation of ether. The more usual procedures, such as resection of rib for empyema, are best performed with the use of C.E. mixture or pure chloroform if there is much lung disease. Where there are cyanosis and shortness of breath the risk of cardiac failure is so great that open ether should be employed as soon as possible after the initial chloroform, in spite of the deleterious effects of the ether on the lung. Oxygen with the anæsthetic is highly desirable in such cases. Position is all-important in empyema, and the patient must

be placed, as far as the surgeon can possibly allow it, on the affected side. *Abdominal* operations are best performed, as a rule, under open ether preceded by atropine ($\frac{1}{10}$ gr.), but it is often advisable to pass through the first two stages of anæsthesia with C.E. mixture, particularly in the case of strong or alcoholic subjects. Gas and ether and similar methods are not recommended unless the operation takes no longer than twenty minutes or so. For all operations below the umbilicus, spinal analgesia gives good results in expert hands. *Rectal* operations require deep ether narcosis. Those of short duration may have gas and ether, the longer ones open ether. Morphia and atropine should generally be used beforehand.

Nitrous oxide (N_2O , Nitrous Monoxide).—"Laughing gas" alone, or with air or oxygen, being the safest known anæsthetic, should be used in all operations lasting up to about a quarter of an hour in which absolute muscular relaxation is not essential. It must not be relied upon alone if an anæsthesia of more than about half a minute is required for cases in which the face-piece has to be removed to allow operation; unless the anæsthetist is expert with the nasal method of administration. It suffices for incising an abscess or whitlow, etc., and for removal of one or a few teeth.

Method of administration.—Whether the patient is lying or sitting, arrange him so that the chest is in its natural relation to the shoulders, the neck being neither flexed nor extended. Insert a small prop between the first incisors, or elsewhere as necessary, in the case of tooth extraction, and carefully fit on the face-piece so that all air leakage round its edge is excluded. Turn the foot key so as to fill the bag about two-thirds full. Instruct the patient to breathe quietly in and out of the mouth. Having seen that the expiratory valve is working, and knowing therefore that the face-piece is fitting accurately, turn the stop-cock so as to admit gas from the bag to the face-piece. It will be inhaled into the lungs, expirations escaping through the expiratory valve into the air. Keep the bag nearly full by frequent gentle turns of the foot key. Breathing becomes deeper and the pulse fuller. After about twenty seconds consciousness is lost. A little later the third stage, surgical anæsthesia, is entered upon. The breathing, which has been rhythmic, becomes irregular and jerky, and the characteristic "stertor" is

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heard. The face is more or less dusky. Clonic muscular contractions are common, and sometimes tonic spasm occurs, resulting in opisthotonos. The conjunctival reflex disappears but not the corneal; the pupils are widely dilated. These symptoms of anæsthesia usually arise after about thirty breaths. The available anæsthesia from nitrous oxide is slightly prolonged if, towards the end of administration, the expiratory valve is closed and rebreathing permitted for a few respirations. It is obvious from what has been said that the anæsthesia of pure nitrous oxide is curtailed by the advent of what are truly asphyxial symptoms, which, if the administration is to be prolonged, must be prevented from arising. This is attained by the use of air or the admixture of oxygen. With either of these measures it is possible to continue administering gas in such a way that operations of ten minutes' duration or more can conveniently be performed, and the anæsthetization can be continued by the nasal route when the operation involves the mouth, as in the extraction of several teeth.

With gas and air, the administration is begun as with nitrous oxide alone. After about ten breaths, one breath of air is permitted by opening the stopcock; then five more breaths of gas and another of air are given, and, when anæsthesia is obtained, breaths of gas two or three at a time are alternated with breaths of air in such a way that the stertor or clonic movements when just arising are immediately checked. In this way the administration of gas and air may be continued for a considerable time. The extent to which air is admitted varies with different individuals; the stronger and the more plethoric the patient, the less can air be used; the more anæmic or weakly, the more it is needed. For *gas and oxygen* special apparatus of a more elaborate nature is required. The use of this and of *continuous nasal gas* is best reserved for specialists or those who have frequent opportunities for employing such measures, descriptions of which must be sought in works on anæsthetics.

Ethyl chloride (C_2H_5Cl).—The use of this anæsthetic is also restricted to short operations or to the introduction of more formidable agents. Ethyl chloride is of extreme value when nitrous oxide is not applicable, and its great portability and the simplicity of the apparatus are great advantages; but its use is not without danger, and therefore it should never replace nitrous oxide except for good reason. In the case of young children, for

example, it is often invaluable; they give a very short period of anæsthesia with nitrous oxide and easily become convulsive.

Method of administration.—Except in infants, for whom it can be used efficiently from a Skinner's mask, ethyl chloride should be given by a process of rebreathing from a closed inhaler. The simplest apparatus consists of the small bag of a Clover's inhaler supplemented by a metal tap through which ethyl chloride can be squirted into the bag; or a small tube may be fitted on to the top and the drug tilted in through this. The metal mount of the bag fits straight on to a face-piece, and this, with a tube of ethyl chloride, completes the necessary outfit. The dosage must be regulated carefully. For a child, squirt 2 or 3 c.c. into the bag; for a woman, 4 c.c.; and for a man, 5 c.c. Fit the face-piece accurately to the face, the bag hanging down. Instruct the patient to breathe quietly and raise the face-piece with the second inspiration, replacing it so as to catch the expiration in the bag. Now gently raise this, the face-piece remaining in close apposition to the face. At the fourth breath give another inspiration of air. By the fifth breath the bag should be held at right angles to the face-piece. The patient is now unconscious, and after two more breaths the operation may be performed, no further anæsthetic being required unless obvious signs of recovery arise before the operator has finished.

The anæsthesia from ethyl chloride is marked by none of the violent respiratory or muscular phenomena seen with nitrous oxide, nor does the colour change greatly, and for these reasons it is not easy for the inexperienced to decide when enough has been given. Fixed globes of the eyes and absent conjunctival reflex are enough to determine the end of the administration; stertor is not always present, and should not be waited for, if the eye signs are already present. A prop should always be placed between the teeth before beginning, for there is often spasm of the jaw muscles.

Ether [$(C_2H_5)_2O$].—Two kinds of ether are available, one made from pure rectified spirit (Ether Off.), and the other from methylated spirit (Rectified Ether). There is little to choose between them, but the second is much the cheaper. Ether may be given (1) by closed methods, (2) by the open method, (3) intravenously, (4) by intratracheal insufflation, (5) by rectal injection, (6) by swallowing. The first two alone will be described in detail, as the other methods concern only the specialist.

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Closed methods.—The invention of Clover's regulating ether inhaler made possible the gradual administration of an increasingly strong ether vapour without great discomfort to the patient. The best form of this instrument is that associated with the name of Hewitt. With this apparatus ether can be given safely and conveniently to the vast majority of persons between the ages of 6 and 60, and its use should therefore be familiar to the practitioner. The procedure is as follows: Put the indicator to " $\frac{1}{4}$," and insert 2 oz. of ether. Turn the indicator back to "0," and, applying the face-piece to your mouth, blow strongly through the inhaler. All smell of ether will thus be driven out. The small bag is now fixed to the top of the inhaler, and the face-piece may be gently but accurately applied to the patient's face. This will be turned to the right side. Hold the face-piece firmly with your left hand and raise it from the chin as the patient inspires for a couple of breaths, catching his expirations in the small bag, which will thus be moderately distended. Now, with the right hand, very slowly turn the indicator away from "0." Regular breathing without cough or holding the breath will show that you are not admitting the ether too fast, and, this being so, steadily push on the indicator till half-way to "full." Give one breath of air and then continue. It should take one minute to reach the " $\frac{1}{4}$ " mark and at least four to arrive at "full." By this time breathing will be stertorous, the face flushed, the pupils moderately dilated, the muscles relaxed, and the conjunctival reflex abolished. The patient is now ready for most operations. For abdominal and rectal operations and those on particularly sensitive parts, as the ends of the fingers or the eyes, a further stage must be reached, marked by abolition of the corneal reflex also. From this time onwards breaths of air are to be given by raising the face-piece sufficiently often to keep the patient free from cyanosis. While inducing the anæsthesia a certain amount of duskiess is justifiable, for too free admission of air will lead to excitement and delay anæsthesia, but when the incision has been made without reflex movements being produced, adequate aeration must be provided throughout. After the initial incision the indicator is put back to " $\frac{1}{4}$." The longer the operation, the less ether will be required, and the indicator may be at " $\frac{1}{4}$ " or even farther back for most of the time in long cases. Coughing and swallowing movements

indicate too light a narcosis, and are to be met by pushing forward the indicator and temporarily diminishing the supply of air. On the other hand, widely dilated pupils with absent corneal reflex, profuse sweating, duskiess or pallor of the face, are signs that too much anæsthetic is being given. Remove the inhaler altogether, open the mouth and swab out the pharynx with a sponge.

Ether is often preceded by *nitrous oxide or ethyl chloride*. The advantages gained are that unconsciousness is produced more speedily and with less unpleasantness to the patient. In the case of *nitrous oxide* the disadvantage is the necessity for considerable additional apparatus—cylinders of gas and the bag with stopcock attached to it (Fig. 1). This is to be fitted to the

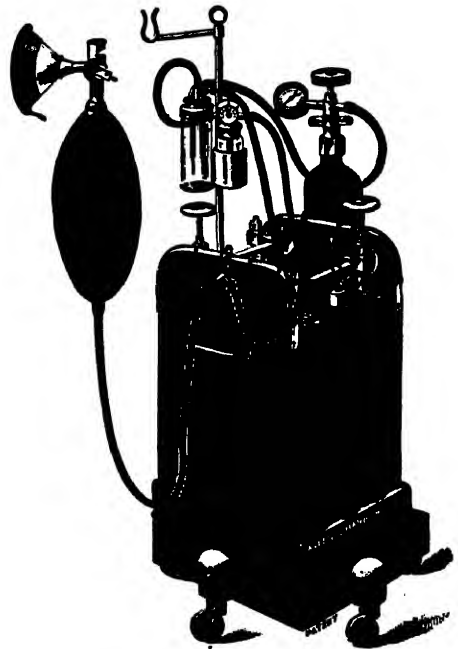


Fig. 1.—Boyle's nitrous-oxide and oxygen-and-ether apparatus.

Clover's inhaler, and the administration begun as for nitrous oxide alone. After three breaths of gas have been inhaled, the stopper is taken from the opening in the inhaler and 2 oz. of ether are introduced. The indicator has till now been at "0," the patient inhaling nitrous oxide and breathing into the air. After six breaths of nitrous oxide, start cautiously turning on the ether, the patient now getting gas with increasing amounts of ether. When the half-

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way point is reached, give a breath of air by means of the stopcock; afterwards more rapidly push the ether till the "full" mark is attained. Then replace the gas bag by the small bag of the Clover's inhaler and continue as in the administration of ether alone. Similarly, with *ethyl chloride* the process consists in the initial induction by ethyl chloride as described, the bag, however, fitting on to the Clover's inhaler instead of directly on to the face-piece. The inhaler is previously loaded with ether, and this is added to the ethyl chloride after the first three breaths, so that the patient breathes in more and more ether as the indicator is advanced. A breath of air at the fourth respiration and again at the eighth should be given, and when the "full" mark is reached the bag should be temporarily removed and emptied of any ethyl chloride that it may still contain; then the administration proceeds as if with ether alone.

Open method.—This is to be preferred for all long operations, and when the patient's condition makes him an unfavourable subject for any air limitation. It is especially advantageous in abdominal operations because of the quieter character of the respiratory movements. The induction takes considerably longer than with closed methods, and, in the case of very strong or alcoholic subjects, the method is not adequate unless supplemented by C.E. mixture or chloroform. The procedure is admirably simple. The patient should always have a hypodermic injection of atropine ($\frac{1}{160}$ gr.) from half to three-quarters of an hour before the operation. Some anæsthetists use, in addition, morphia ($\frac{1}{4}$ gr.), or scopolamine ($\frac{1}{160}$ gr.), or both. When these drugs are employed the patient should not walk to the operating table. The face being guarded around the mouth and nose by a thick pad of bandaging or some turns of gauze, a metal mask of the Skinner or Schimmelbusch pattern, covered with ten layers of fine surgical gauze or two of domett, is placed upon the face pad. The patient breathes freely through the mask covering, on which drops of ether are allowed to fall in a slow stream. A bottle must be employed which holds at least 8 oz. and permits the ether to escape by single drops. At first these are allowed to flow so slowly that only about one per breath is inhaled. Gradually the larynx becomes accustomed to the irritating vapour, and the rate of dropping is increased till in about five minutes the whole surface of the mask is kept moistened. The dropping must

be maintained steadily so that the inhalation of ether vapour is continuous throughout the administration, for only in this way can efficient narcosis be maintained. The face becomes flushed and the pulse and respiration are stimulated, but to a less extent than with closed methods; indeed, in feeble subjects the character of the breathing is more comparable with that during chloroform anæsthesia. The liability to shock and to reflex disturbances of breathing is, however, much less. In average adult subjects anæsthesia is established in about eight minutes. Sweating, salivation, and secretion of mucus are considerably less than with closed methods, even if no atropine is used beforehand.

For operations within the *mouth*, removal of the upper jaw, etc., Crile's tubes may be used. These connect with a funnel on the covering of which ether is dropped, and are themselves passed into the naso-pharynx, gauze being carefully packed around them there. Anæsthesia is first secured in the ordinary way, and the pharynx is then freely swabbed with 5-per-cent. cocaine solution. For these cases,



Fig. 2.—Shipway's warm vapour apparatus for ether and chloroform.

and still more for *intrathoracic* operations, the method of *intratracheal insufflation* is advantageous. This method and that of *intravenous infusion of ether*, a process which is valuable in cases of shock, require special apparatus and considerable practice, and are beyond the scope of this article. Mention must be made, however, of Shipway's warm vapour apparatus (Fig. 2),

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an excellent contrivance for the giving of ether vapour, which can be used whenever the open method is applicable. The apparatus can deliver chloroform also, and may therefore replace the Junker apparatus. Like the latter, it is worked by a rubber-ball pump or by a stream of oxygen from a cylinder.

The **rectal administration of ether** also has advantages in prolonged mouth and nose cases. The colon must be well irrigated three hours before the administration. One hour before operation a preliminary dose of ether and olive oil (4 dr. of each) containing 5 gr. of chlorotone is inserted into the rectum. A quarter of an hour later, morphia ($\frac{1}{4}$ gr.) and atropine ($\frac{1}{16}$ gr.) are given hypodermically. Before operation, a mixture of 6 oz. of ether in 2 oz. of olive oil is poured slowly through a tube into the rectum at the rate of 1 oz. a minute. The maximum amount to be inserted is 8 oz., the quantity required being regulated by the formula—1 oz. of the mixture for every 20 lb. of body weight of the patient. For children the mixture consists of equal amounts of olive oil and ether. Anæsthesia is reached in 10–30 minutes. Cyanosis, loss of reflexes, or embarrassed respiration is an indication for withdrawing 2 or 3 oz. of the fluid. At the conclusion of the operation the rectum is washed out with soapsuds and water through the rubber tubes.

Chloroform (CHCl_3).—Being applicable to all cases, and capable of being administered with the simplest of apparatus, chloroform retains a prominent position as a general anæsthetic; yet its great convenience should never allow us to overlook the fact that it possesses dangers absent from the other drugs with which we have to deal. These dangers depend on the intrinsic toxic properties of chloroform, and, although the exact physiological explanation offered varies with different authorities, the clinical fact remains that fatalities will occur if chloroform is used as a routine anæsthetic. These fatalities are not all due to the administration of an overdose—i.e. a too high percentage-vapour—of the drug, nor to obstructive asphyxial difficulties arising in the course of anæsthesia. It may be that ventricular fibrillation of the heart, which has recently been described, may be the real *fons et origo mali*. Fatalities difficult to prevent may occur during the early stages of chloroform inhalation, as well as others more preventable which arise during full anæsthesia and are probably to be accounted for by the inhalation of too strong

a vapour. The first practical lesson to be drawn from experience, both physiological and clinical, is that anæsthesia should never be induced with chloroform alone if it is possible to avoid it. The second is that the chloroform should be so freely diluted with air that the vapour inhaled has no higher a percentage than 2 parts of chloroform to 98 of air. To achieve this second desideratum with scientific accuracy much apparatus has been designed. This is of two kinds, the *plenum* variety and the *draw-over* kind.

The **plenum apparatus**, though capable of the greatest accuracy, has the disadvantage that it requires motor power for driving a current of air through or over the chloroform. Consequently, these machines are too cumbersome for use except in hospitals or similar institutions. The Dubois, the Roth-Dräger and Waller's balance are the best-known examples. The only apparatus, on this principle, which is in common use in Great Britain is *Junker's chloroform inhaler*, an extremely serviceable instrument in spite of the drawback that it is impossible to be sure of the strength of the vapour inhaled from it. Yet it is so constructed that the vapour pumped from it is within the safe limits of about 3 per cent. It is of especial use when fitted with a tube and employed for continued anæsthesia in nose and throat operations. The anæsthetist must see that it is in good working order, and that the exit and entry tubes within the bottle are properly adjusted to the mask or tube and to the pump respectively. At the beginning of the administration air is pumped very gently through the ounce of chloroform in the bottle by means of gentle compressions of the hand pump. Each squeeze is given so as to correspond with the inspirations, the mask being held lightly on the face. After about a minute, if the breathing is uninterrupted, the squeezes are made more powerful till, after three or four minutes, with every inspiration the rubber ball is being compressed to almost one-fourth of its volume.

The only **draw-over apparatus** in use is *Vernon Harcourt's inhaler* (Fig. 3). This provides a mixture of air and chloroform automatically limited to a maximum strength of 2 per cent., which can be diluted at will to any smaller proportion and increased by a special arrangement to 2·5 or 3 per cent. The face-piece has to be fitted accurately to the patient's face and the instrument must be kept perfectly steady. To ensure this a long tube is provided, permitting the chloroform and containing part of the

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apparatus to be fixed immovably at a distance from the face-piece. The current through the apparatus depends upon the patient's respiration. Induction is slow, and with strong

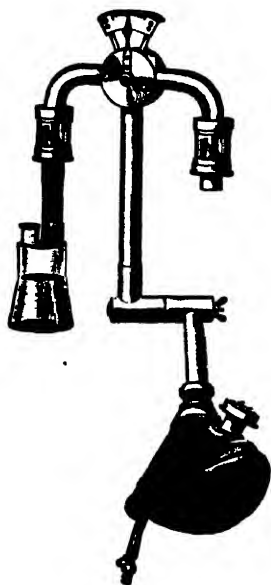


Fig. 3.—Vernon Harcourt's chloroform inhaler.

subjects no adequate anæsthesia for all operations can be guaranteed.

The most generally useful method of giving chloroform is by means of a **drop-bottle and Skinner's mask**. Without care and experience, however, danger easily arises. The governing principle is to give the chloroform gradually, continuously, and always plentifully diluted with air. This is attained by beginning with drops at the rate of one per breath, by moistening the whole surface of the mask only by degrees, and by *never letting the mask rest upon the patient's face*. By this means there is always a layer of air between mask and face, and free dilution of the evaporating chloroform is ensured. The mask should not be covered by more than a single layer of flannel or domett. The drop-bottles of the *Hewitt* and *Thomas* patterns are perhaps the best.

Four stages of chloroform anæsthesia may often roughly be distinguished. They are: (1) *A stage of analgesia*, distinguished by disturbances of judgment, control, and sensation; rapid flight of ideas; emotionalism and dreams; pupils dilated; reflexes well marked; respiration increased with sometimes cough and hold-

ing of breath (2) *A stage of complete loss of consciousness*, with muscular spasm, smaller pupils, and deep respiration, the reflexes still present. (3) *A stage of surgical anæsthesia*, with relaxed muscles, absent conjunctival reflex, regular breathing, and larger pupils. Later the corneal, laryngeal, pharyngeal, and patellar reflexes are absent. (4) *A stage of bulbar paralysis*, with loss of rectal and bladder reflexes; dilated, fixed pupils; separated eyelids; and death.

Chloroform mixtures.—The desire to avoid the adverse effect of chloroform upon blood-pressure and the musculature of the heart, lowering the first and paralyzing the second, led to the use of mixtures containing, as well as chloroform, subsidiary drugs which act as correctives to its depressant action. These mixtures are a compromise, and are less safe than ether but not so dangerous as chloroform alone. They are very useful for those who have not frequent opportunities of giving anæsthetics, for they can be employed in every variety of case. Various proportions of ether and of chloroform are used. We need here mention only two of the best-known—(1) the *A.C.E. mixture*, containing alcohol 1 part, ether 2 parts, chloroform 3 parts (by volume); (2) the *C.E. mixture*, composed of chloroform 2 parts and ether 3 parts (by volume). The alcohol was originally included to equalize the evaporation of the ether and chloroform. Administered in the way in which we now believe these mixtures should alone be used—viz. by the repeated additions of small quantities on the open mask—the question of unequal evaporation becomes of no practical account, for the whole of each small amount is evaporated and inhaled as it is offered. I therefore recommend the C.E. mixture as the best all-round example of this kind of anæsthetic. While employing it, the anæsthetist must bear in mind the principles which guide him in giving chloroform, and must act as if he were administering chloroform alone. Consequently, he will use the same apparatus—mask and drop-bottle—and the same general procedure, i.e. gradual administration and free air admixture. The latter, however, is practised with less rigour. For instance, in the case of strong or alcoholic subjects two layers of domett may be used on the mask, and a gauze pad for the face, as in the case of open ether. Moreover, the mask may be kept saturated with frequent additions of C.E. mixture in a way which is never permissible with pure chloroform. The

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mixture is advisable in the case of obese, plethoric, and bronchitic subjects who do not respond well to ether alone and whose cardiac condition makes the use of pure chloroform inadvisable.

Narcotic alkaloids.—The use of alkaloids for narcotic purposes may briefly be discussed at this point. We have seen that in the case of open ether a preliminary injection of *atropine* is regarded as an essential part of the method. There is much to be said for its employment as a routine measure before all abdominal operations, no matter what anæsthetic be used. Diminution of shock and of after-sickness is a good reason for this practice. With regard to *morphine* and *scopolamine* the question is not so simple. Some persons are nauseated, even made faint or delirious by these drugs, so that, unless the personal idiosyncrasy of the patient to them is known beforehand, they cannot be used indiscriminately without risk. It is possible to rely on repeated injections to produce anæsthesia, as is largely done on the Continent. For ordinary surgical operations, however, this anæsthesia is not to be preferred to that obtained by general anæsthetics. For *obstetric purposes* there is much to be said for the use of *scopolamine* in this way, for it allows anæsthetization just when the patient most needs it, viz. in the early stages of labour. The infant at birth, however, is often detrimentally affected by the narcotic and may require prolonged assistance before respiration is established properly. In the case of nervous and of ultra-robust or alcoholic patients, the hypodermic injection of *morphine* ($\frac{1}{4}$ gr.), *scopolamine* ($\frac{1}{10}$ gr.), and *atropine* ($\frac{1}{10}$ gr.) before prolonged operations gives excellent results.

Local anæsthesia.—A knowledge of the use of local analgesics is of the greatest value to the practitioner, for it enables him to perform safely and conveniently a number of operations without the assistance of a second medical man. Apart from this consideration, it is often highly desirable to be able to perform an operation without the administration of a general anæsthetic. Broadly speaking, superficial operations, the removal of small tumours, operations for hernia and similar procedures are alone satisfactorily performed with the use of local analgesics. They are particularly useful in the case of strangulated hernia when the condition has existed for some time, and regurgitant vomiting adds to the danger of a general anæsthetic. The patients, especially

when the hernia is umbilical, are often extremely fat and, because of this and of poor lung expansion, unfavourable subjects for general anæsthesia.

Since the introduction of *cocaine* the use of local analgesics has made great strides. This drug is now largely replaced by others of less poisonous nature except for painting upon mucous surfaces or instilling into the eye. For the latter purposes it is used in strengths of 2–10 per cent. For injection, the solution should not be stronger than 1 per cent., and the amount injected should not be more than 1 gr.; if the solution is still more dilute, the amount of cocaine permissible is also greater. Some persons are extraordinarily sensitive to the action of cocaine, and poisoning symptoms have been produced by "safe" amounts, injected, for instance, for excision of external piles. The symptoms of *cocaine poisoning* are those of collapse and of mental excitement. In mild cases the patient becomes talkative and emotional; in those more severe, maniacal and delirious. Muscular tremor followed by convulsions may occur; more often there are pallor, perspiration, nausea, feeble breathing, and syncope. The treatment is to place the patient in the horizontal position and administer stimulants such as strychnine and hot coffee. There is no special antidote. Artificial respiration may be necessary.

Cocaine has been largely superseded by *eucaine*, and the latter, in turn, by *novocain*. This drug and *allypin* have, broadly speaking, banished cocaine from the realms of ordinary surgery so far as local analgesia is concerned. Their action is enhanced by the addition of *adrenalin*, and they are commonly used in conjunction with a 1-in-1,000 solution of this drug. The ischæmia produced by the adrenalin localizes and intensifies the action of the analgesic. Not more than about 15 drops at a time of the adrenalin solution should be used, but they may be freely diluted. *Adrenalin* also prolongs the action of *novocain*, which is apt to be too evanescent. *Novocain* is free from toxic and irritating properties, and may consequently be used in large amounts. The 2-per-cent. solution is most commonly employed.

Local analgesics are used according to two methods: (1) *infiltration anæsthesia*, (2) *regional anæsthesia*. Infiltration anæsthesia, which has the widest application, is the method by which the analgesic is injected directly into

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the tissues to be operated upon; in regional anæsthesia the drugs are injected into or around the nerves supplying the operation area. The technique of local anæsthesia must be carried out with the strict surgical cleanliness observed in other operative measures. The skin is prepared and the needles and syringes are sterilized in the usual manner. When boiling the latter, care must be taken that no soda is used in the water, for it decomposes some of the

the analgesic, they must be infiltrated with it. All pulling and tearing of tissue must be avoided or pain is caused outside the infiltrated area. Owing to the adrenalin, bleeding is slight, but, as reactionary hæmorrhage may occur, any point which bleeds during the operation must be ligatured and pressure must be firmly applied by the dressing.

2. **Regional analgesia** is highly successful for parts the nerves to which are readily accessible.

Thus, for operations on the hand or foot a sure and simple method is to infiltrate completely a layer of tissue bounding the field of operation on its proximal side. In the forearm and leg the ulnar, musculo-spiral, median, tibial, and popliteal nerves, being accessible, can be anæsthetized at definite points by injecting strong solutions around them. Analgesia takes about 10-15 minutes to develop. Obviously, an accurate knowledge of the anatomy of the nerve supply of the part is necessary for successful application of the method. In ordinary practice, operations upon the fingers and toes provide the chief field for regional analgesia. The skin at the base of the digit is pinched up from the side, and the needle passed into the subcutaneous tissue on the dorsal aspect just

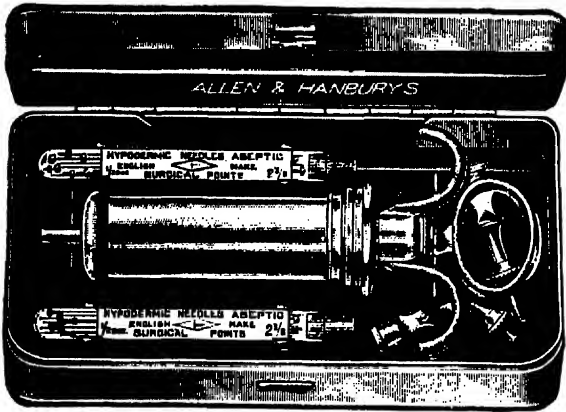


Fig. 4.—Gray's syringe and needles for local analgesia.

local anæsthetics. The syringe should hold at least 2 c.c.; the "Record" is a good type, commonly used. Needles must be sharp-pointed and firm. (Fig. 4.)

1. **Infiltration anæsthesia** is thus carried out. First, a series of small injections are made into the dermis, not subcutaneously, along the line of incision. Each injection raises a white wheal, on the edge of which the next injection is made. A band of analgesia being thus produced, the incision is made, and succeeding layers of tissue are infiltrated as they are exposed. It is necessary to incise exactly in the line of the injections and to inject exactly along the line of the proposed incision. Similarly, in inserting stitches the needle must not be put in beyond the narrow analgesic band. The method requires great care and precision. Generally speaking, about 3 drachms are required to inject a 3-in. incision, a slight but uniform swelling being produced over the whole area of injection. Considerable time is necessary for the development of analgesia, and this is one of the chief disadvantages of the method. Twenty minutes may elapse after injection before the part is thoroughly insensitive; and it is of no avail merely to bathe the tissues in

to one side of the phalanx. It is then pushed on till the point lies near the skin on the palmar aspect. About 10 drops are then injected, and another 10 as the needle is slowly withdrawn. In this way a semicircular solution is made in the subcutaneous tissue round one half of the finger. The process is repeated round the other half, and then the whole digit distal to the injections is insensitive after 10 minutes or so. (For details of injection of larger nerve-trunks the reader is referred to larger works on local anæsthesia.)

Spinal anæsthesia.—This form of anæsthesia is of service for all operations below the umbilicus when a general anæsthetic is inadvisable. Such instances are common in connexion with diabetes, severe cardiac and pulmonary disease, gross damage to the lower extremities accompanied by shock, and also in advanced septic disease in the abdomen in children. Apart from these instances, spinal analgesia may be preferred in ordinary work if the patient is anxious for its use and if the surgeon is thoroughly accustomed to it. In such circumstances it may be regarded as less safe than ether but not more dangerous than chloroform. The chief drugs used for spinal

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analgesia are *stovaine* and *novocain*. An excellent and much-used injection is that of Barker, *stovaine* 5 parts, glucose 5 parts, distilled water 90 parts. This fluid is isotonic with the blood and its weight enables one to localize its action by gravity. One c.c. of the solution contains 5 cg. of *stovaine*, which is a customary dose.

To make the injection, place the patient with the back well arched either in a sitting posture or, if it is not advisable for him to sit, lying on one side with the knees drawn up to the chest. By defining the highest points of the iliac crest on each side, a line joining which will cross the fourth lumbar spine, the interspace for injection between this and the third lumbar spine is easily found. The hands having been carefully disinfected, a finger is placed upon the fourth spine and the trocar entered in the middle line just above the finger. It is directed slightly upwards and enters the theca at a depth of about 8 cm. If only blood issues, no injection is made but another puncture tried. If cerebro-spinal fluid tinged with blood or pure cerebro-spinal fluid escapes, the syringe is fitted to the trocar and the injection made after about as much fluid has escaped as it is proposed to inject. The trocar is now withdrawn and the puncture sealed with sterile gauze and collodion. The patient is then laid on his back with a pillow beneath the head and shoulders. If the injection is made with the patient lying on one side, the side must be that of the limb to be operated on, if the operation is upon a lower limb. If the operation is on the abdomen the pelvis should be raised with a pillow as well as the head, so that the dorso-lumbar spine is the lowest part of the trunk. In about five minutes the knee-jerks are lost, and this is followed by gradually increasing analgesia of feet and legs, thighs, perineum and abdomen, and later by loss of motor power in the same regions. Subjectively there are sensations of warmth, swelling, and heaviness in the lower limbs. Frequently, about a quarter of an hour after injection, there are pallor, nausea, sweating, feeble pulse, and vomiting, symptoms attributed to the effect of splanchnic dilatation resulting from feeble breathing. Generally they rapidly pass off. Not infrequently the sphincter ani is relaxed. Anæsthesia lasts for about an hour. Headache and pyrexia are the most frequent after-effects. Postoperative pneumonia has occurred fairly often. Nervous paralysees have been reported in a small num-

ber of cases. Probably, failure to anæsthetize is due to errors of technique, the trocar not having entered the theca.

Treatment of danger.—Symptoms of danger seen during operation are complex, arising both from the work of the operator and from the administration by the anæsthetist; yet it is the peculiar duty of the latter to look out for them, to warn the operator so far as his performances are responsible, and to institute the proper treatment. The character of the respiration is the main indication for the anæsthetist throughout and, in conjunction with the colour of the face and the condition of the pupils, always guides aright if constantly observed. Suppose that respirations have become feeble, the face is pale, the pupils are dilated, and the corneal reflex is absent. The condition may be due to surgical shock or to overdose of anæsthetic. If there is nothing in the operation to warrant surgical shock—no great loss of blood, no handling of sensitive structures—the anæsthetic must be held responsible. The measures to take are these:

1. Remove the anæsthetic.
2. Briskly rub the face with the towel.
3. Lower the head.
4. Partly open the mouth with Mason's gag and gently draw forward the tongue after passing a sponge rapidly round the back of the mouth. In all mild cases these measures will at once restore the breathing and, secondarily, the circulation.

If these measures are not successful within a moment or two and the respiration ceases—

5. Firmly compress the chest by placing a hand on each side of it and bringing your weight to bear forcibly upon it. If after three compressions spontaneous breathing is not restored, then perform artificial respiration by Sylvester's method (see RESPIRATION, ARTIFICIAL).

When artificial respiration is necessary the anæsthetist should carry it out, handing over to a nurse or an assistant the duty of keeping the mouth open and the tongue held lightly forward. Similarly, an assistant may be asked to make a hypodermic injection of strychnine if the condition has developed suddenly and is thought to be due primarily to reflex circulatory failure. It is to be borne most prominently in mind that the simple measures of lowering the head, establishment of a free

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airway, and stimulating the respiration, are the most important, and no injection, infusion, etc., should be allowed to prevent their immediate performance.

J. BLOMFELD.

ANAL FISSURE.—A very painful and sensitive ulcer of the anal margin. It is usually situated posteriorly and is triangular in shape. The narrow base of the ulcer is at the anal margin, and is often hidden by a small tag of skin or external "sentinel pile" which indicates the exact site of the lesion.

Etiology.—Painful anal fissure is nearly always due to tearing of the anal margin by hard scybalous masses. Most likely a valve of Morgagni is torn and forms the external tag so commonly seen.

Symptomatology.—The patient complains of severe pain after defæcation, lasting a long time—half an hour to three hours—and felt not only in the anal region but also in the thighs and buttocks. The movements of the bowels are so painful that the patient dreads an action, and consequently the initial constipation becomes worse. Spasm of the external sphincter is a fairly constant accompaniment. By separating the margins of the anus the ulcer can be seen. Long-standing fissures often have a hard fibrous base. Anal abscesses and small fistulæ may develop.

Diagnosis.—Anal fissure can almost always be diagnosed by the history alone. The account of excruciating pain lasting for some hours after defæcation is sufficient to indicate the condition, while local examination will often show the small external pile or tag of skin at the base of the ulcer. Owing to spasm of the external sphincter the anal margin is drawn up and the edges of the anus have to be everted carefully for the ulcer to be visible. There is no need to insert the finger in the rectum in order to diagnose anal fissure.

There is a type of anal fissure sometimes found in patients suffering from syphilis in which the ulcers may be found anywhere around the anal margin; these are often multiple, and almost painless.

Treatment.—The first essential is to get the bowels to act easily and well by the regular and careful use of laxatives. The pain may be relieved by the local application of an ointment containing 4 gr. of cocaine to 1 oz. of vaselin. Healing may sometimes be promoted by painting the ulcer with silver nitrate solution (5 or 10 gr. to the ounce) after anæsthetizing the surface by applying a

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pledget of wool or gauze soaked in 5-per-cent. novocain.

If the fissure shows no tendency to heal, the base of the ulcer should be divided by a sharp blunt-pointed bistoury. For this purpose local anæsthesia often suffices. Half to one drachm of a 1-per-cent. solution of novocain should be injected through a fine needle under the area occupied by the ulcer and the external pile before the incision is made. It is necessary to divide some of the fibres of the external sphincter under the base of the ulcer. In sensitive patients and in those with long-standing and fibrous fissures it is well to give a general anæsthetic and, after thoroughly exposing the ulcer, either to excise it completely, or divide its base and most of the underlying sphincter. Stretching the sphincter is not to be advised, since it causes much more shock than cutting. Any external pile is to be cut away at the same time.

ZACHARY COPE.

ANAL FISTULA.—A sinus lined by granulation tissue which persists after the occurrence of an ischio-rectal abscess (*see* ABSCESS, ISCHIO-RECTAL). If the abscess bursts, or is opened, through the skin only, a blind external fistula results. When the abscess also opens into the anal canal a complete fistula in ano is left. Occasionally the abscess-track communicates with the anal canal but not with the exterior through the skin, thus forming a blind internal fistula. Sometimes the sinus tracks upward beneath the rectal mucous membrane, or even rarely through the levator ani muscle, while frequently it burrows outward into the buttock or round the margin of the anal sphincter.

If a transverse line be drawn across the middle of the anal opening, external sinuses in front of the line will be found to open into the anal canal immediately opposite the external opening, while all fistulæ posterior to that line have their internal opening in the mid-line posteriorly. Small fistulæ secondary to anal abscess may form an exception to the rule. The internal opening usually lies between the external and internal sphincters.

Etiology.—The precursor of a fistula is either a septic or a tuberculous ischio-rectal, anal, or pelvi-rectal abscess. The track persists because the original opening made into the abscess is insufficient to provide free drainage, the cavity is allowed to close up too soon, or the wall of the sinus is infiltrated with

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tuberculous material. If it persists for a considerable time the formation of dense fibrous tissue makes it very difficult for blood to obtain access to the focus of infection, and thus healing is further hindered.

Symptomatology.—The chief and often the only symptom is a discharge from the anus or from the anal region. Sometimes slight pain may be complained of. The discharge varies from thick pus to sero-pus or a serous fluid. Occasionally it may cease, only to come on again more copiously when the sinus reopens. Examination generally shows one or more openings about the anus. By exploring with a probe, the extent of the track can be ascertained and any opening into the bowel discovered. The internal opening can often be felt as a dimple in the mid-line posteriorly. When no external opening can be seen (blind internal fistula), digital or proctoscopic examination will reveal the internal sinus from which the discharge is coming.

Diagnosis.—The only difficulties in diagnosis are :

(a) *In those cases in which there is no external opening.*—The occurrence of a discharge, the history of acute pain corresponding to an acute abscess, and careful digital and proctoscopic examination of the anal canal should enable one to make the diagnosis.

(b) *To distinguish from a urinary fistula.*—Generally urinary fistulae are farther forwards in the perineum, and urine comes away from the sinus. Moreover, on trying to pass a catheter or bougie, a stricture of the urethra will be discovered. It must be recollected, however, that urinary fistulae may have openings far removed from the urethra, and anal fistulae sometimes track a considerable distance.

(c) *To distinguish between tuberculous and septic fistulae.*—This is done by paying attention to the history, for a tuberculous fistula does not begin with the acute symptoms of a septic fistula; by examining the patient carefully for tuberculous foci elsewhere (e.g. in lungs, glands, abdomen); and by noting the character of the sinus-opening, which in tuberculous cases may be without much induration, and with edges of a bluish-red colour due to tuberculous infiltration.

Treatment.—Preventive treatment by thorough drainage of all ischio-rectal and anal abscesses is most important. The patient should be made to rest (in bed if possible) until the abscess is properly healed.

Operative interference is almost always

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needed in those cases which have not healed in two or three months. Some few external fistulae may be induced to heal by painting the interior of the sinus with pure carbolic acid. It is well not to put off operation too long, since the tracks may burrow. Operation consists in opening up the whole track of the sinus. It is essential to cut through the external sphincter in all cases of complete, and in some of incomplete, fistulae.

In tuberculous cases the propriety of operating depends upon the presence or extent of tuberculous disease elsewhere.

ZACHARY COPE.

ANAL GROWTHS.—Among simple growths, *papillomata* may occur round the anal margin. The small tags of redundant skin around the anus may become pedunculated. A *polypus* may be formed in the anal canal by the hypertrophy and fibrosis of an internal pile. Small adenomata of the glands in the mucous membrane of the lower part of the rectum often occur in children and may acquire a long pedicle and protrude through the anus on defaecation. They are red, and look something like a dark cherry. Polypi can easily be dealt with by tight ligature of the base.

Malignant growths.—*Epithelioma* may occur as a firm raised papule which ulcerates and gradually extends. The inguinal glands become enlarged. *Melanotic sarcoma* arises but rarely in the pigmented skin round the anus. It forms a dark purplish swelling which in the early stages is likely to be mistaken for a small naevus. The groin glands are sometimes involved.

New growths round the anal margin must be distinguished from the common soft, sodden condylomata of secondary syphilis and from a primary chancre, which is very rarely seen in this situation.

ZACHARY COPE.

ANAPHYLAXIS (see IMMUNITY).

ANARTHRIA (see SPEECH, DISTURBANCES OF).

ANASARCA (see OEDEMA).

ANEURYSM.—A tumour containing blood and arising from one of the arteries of the body.

Etiology.—The essential cause of an aneurysm consists in the giving way of the arterial wall before the pressure transmitted by the blood from the heart. The resulting dis-

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tion may be a purely local bulging, when the aneurysm is called *saccular*, or it may be more general, resulting in a *fusiform* swelling. The arteries are muscular and elastic tubes which are distended with each beat of the heart, but owing to their perfect elasticity they regain completely their original shape in the intervals. With old age and a variety of other causes, toxic and inflammatory, the muscle and elastic fibres may degenerate and be replaced by fibrous tissue, with the result that the tube loses much of its elasticity, but, as a rule, becomes more resistant to the distending forces. Owing to the loss of resiliency the artery, after distension, does not quite return to its original calibre, and in consequence in advanced life the vessels are not only rigid but are also enlarged; a fusiform aneurysm is merely an exaggerated local expression of this general senile dilatation and, hence, is usually found in the aged with very degenerate arteries. Arterial degeneration is often nodular in its distribution, yet, in spite of this, it is very rarely followed by the local bulgings which form saccular aneurysms. The dilatation of a fusiform aneurysm, however, is often irregular in shape through the arterial walls having given way more in some places than in others. No doubt the non-formation of saccular swellings is due to the facts that repair takes place as fast as degeneration, and that the resultant fibrous tissue offers a stronger resistance to distension than even muscle or elastic fibres. In small vessels with poor external support, such as those of the brain, a combination of high blood-pressure and nodular arterial degeneration leads, not uncommonly, to the formation of small saccular aneurysms as well as to rupture; but this is seldom the case with the larger tubes.

It would be expected that in the more distinctly inflammatory lesions the processes of repair would be delayed to a large extent, until the active inflammation was over, and that even then they would be less conspicuous. It is therefore not surprising to find that a local inflammation in the vessel-wall produces a weakened area which may give way to form a local bulging. Thus, when the inflammatory lesions of tuberculosis involve a vessel from the outside, *miliary aneurysms* may form at the weak spots. In this way are explained the miliary aneurysms which are so frequently found in the lungs in fatal cases of hæmoptysis. Inflammation may be excited from the inside

of a vessel by an infective embolus plugging it, and sometimes an aneurysmal swelling arises at the point of obstruction.

The disease which most frequently gives rise to inflammation of the arterial walls is syphilis. In all the stages of syphilis the arteries are more or less affected. Though this is usually by a local endarteritis involving only the inner coat, mesarteritis is common in the tertiary stage, and takes the form of an inflammatory patch which destroys both the muscle and elastic fibres. It is of interest that spirochætes have been found in these patches. This syphilitic mesarteritis is the usual precursor of the sacculated aneurysms of the aorta or other large vessels, and most observers agree that at least 70 per cent. of the cases are syphilitic, the present tendency being to raise this percentage considerably. Other infections also lead to arterial inflammation, as, for instance, enteric fever, rheumatism, or small-pox.

The intima is usually the only layer affected, and the vascular wall is not seriously weakened. Sometimes the intima splits and an opening is established from the lumen so that blood enters between the layers of the arterial wall, forming what is known as a *dissecting aneurysm*. A dissecting aneurysm may similarly arise from the rupture of an atheromatous patch into the lumen.

While syphilis is the fundamental cause of most saccular aneurysms, another factor contributes. Aneurysm occurs much more commonly in men than in women (8-1), and among men it is found to be most frequent in those who lead laborious lives, where much strain is put periodically on the vascular system. No doubt it is in this class that syphilis is most common, but, evidently, the recurring vascular strain plays a considerable part in the causation. It may be that it is in these circumstances that syphilis is most likely to attack the middle coat, or, on the other hand, that among individuals with syphilitic mesarteritis it is chiefly those who put an extra strain on their vessels whose vessel walls give way. It is among the active hard-working labourers, who have in the past had syphilis but are still in the full enjoyment of their muscular powers, that one meets with most of the cases of aneurysm. The commonest age of incidence is between 40 and 50, much earlier than the usual period for any decided evidence of senile degeneration. Though saccular aneurysms are sometimes multiple, it is remarkable

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how healthy the rest of the vessels are in many of the patients.

Aneurysms are prone to occur in situations where the vessel is exposed to most strain and is least effectively supported by outside structures, for instance in the aorta, the great vessels of the neck and, when in the limbs, at the flexures of the joints. No doubt degeneration also specially affects arteries at the points of greatest strain; thus degenerative changes are found most frequently in the aorta and at the places of division of the great vessels. Aneurysm is commonest in the aorta (especially the thoracic), next in the popliteal, and is less common in the great vessels of the trunk, such as the innominate, carotids, subclavians, and iliacs. It is reputed to be more frequent in England and the United States (especially among negroes) than elsewhere, but cannot be described as a very common disease.

Pathology.—Many of the essential points in the structure of an aneurysm have already been discussed. A division used to be made between a true and a false aneurysm, the wall of the former consisting, in part at any rate, of the vascular linings, while the latter was merely an effusion of blood into the tissues from the rupture of a vessel produced either by disease or injury. Such an effusion may be circumscribed, owing to the development of a capsule around it; or diffuse, with no containing wall. A fusiform aneurysm is completely surrounded by the distended, though degenerate, arterial walls, and in very small saccular aneurysms also it is possible that all the layers of the vessel wall may be found in the sac. It is probable, however, that a saccular aneurysm usually begins by a splitting of the tunica intima over a patch of syphilitic mesarteritis, followed by a giving way of the other coats. Such transverse splits of the intima have been found apart from aneurysm in inflammatory conditions of the artery, and have sometimes led, as previously explained, to the formation of dissecting aneurysms. They are not uncommon in mesoarteritis of syphilitic origin, and are not always followed by a further yielding of the weakened wall. The wall of an aneurysmal sac will therefore, as a rule, only show traces of the inner coat at the edges of the opening, which may be small or large, and but little will remain of the diseased middle coat, the main portion of the covering of the sac being formed of fibrous tissue, part of which is derived from the external coat and

part is a new formation produced by the pressure of the tumour on the surrounding tissues. The inner surface of the sac is generally lined by white bloodclot deposited in laminae, but this layer of clot may be quite absent. When present it is variable in thickness, and may even completely obliterate the cavity. If the cavity becomes filled, the central portion of the clot may be formed more quickly and be red in colour.

As the swelling increases in size, it presses on the neighbouring organs, which are liable to become incorporated, so as to form part of the wall of the sac. Owing to the repeated impact of the blood against them they are often destroyed to some extent, and thus vessels may be occluded, nerves irritated or functionally incapacitated, bones eaten away, openings established into the cavities of the body or internal tubes, or the aneurysm may even force its way to the surface and burst externally. Movable organs, such as the heart, may be very considerably displaced, while trophic changes may take place in the lungs either from direct pressure or from the retention of secretions when a bronchus is obstructed. Pressure on a vascular organ often produces congestion; thus there may be congestion of a portion of the pulmonary tissue or of the mucous membrane of the air-passages, oesophagus, or stomach, and this is liable to be followed by bleeding, with the production of hæmoptysis, hæmatemesis, or melaena. Such congested patches are prone to ulcerate, and ultimately a communication may be established with the cavity of the aneurysm itself and may permit the escape of blood from it. From a mucous surface the hæmorrhage is generally slight and intermittent at first, the aneurysm being said to weep, the hole, in the intervals, being closed by clot. As a rule, this ultimately gives way and the patient dies suddenly from loss of blood, but several cases are on record in which an aneurysm, after weeping, has been permanently sealed up by clot.

Pressure on some part of the respiratory apparatus is almost certain to occur when there is an aneurysm within the confined cage of the thorax. The lungs may be affected in various ways. Direct pressure upon a lung may occur, a portion of it collapsing while another part becomes congested. A bronchus leading to a part or the whole of one lung may be either completely or partially occluded. When the obstruction is complete the lung

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collapses and remains atelectatic. With only partial occlusion there is a tendency for the bronchial secretion to be retained and become septic, with the natural sequel that the walls of the bronchioles ulcerate and form bronchiectatic cavities while the surrounding lung becomes fibrosed.

If left alone about 40 per cent. of aneurysms burst. The rupture may take place into the surrounding tissues, the aneurysm becoming diffuse. Such a rupture is not of necessity immediately or even ultimately fatal, though usually death follows very soon. The rent into a serous cavity, such as the pleura, the pericardium, the peritoneum, or a joint, is usually a large one, so that death is generally instantaneous, while in the case of a mucous surface such as that of the trachea, bronchi, or oesophagus there is frequently weeping for some time before the final fatal escape of blood. External rupture is rare; it follows ulceration of the skin in front of an advancing tumour. It is remarkable that an aneurysm, after weeping on the body surface, has been observed to be permanently closed by bloodclot.

It is possible, though the event is rare, for an aneurysm to be spontaneously cured by the formation of clot within it. Obliteration of the sac by clotting may also result from medical or surgical treatment.

When the thrombus formation is slight, aneurysms tend to grow steadily larger and sometimes become enormous, without rupturing or without causing death by pressure on the viscera.

Apart from rupture, aneurysm may cause death in a variety of ways. If situated in the thorax it may press upon the air-passages, or the patient may be worn out by excessive pain and breathlessness. Embolism may follow detachment of some of the clot, and, in the limbs, gangrene may result from interference with the blood supply. Abdominal aneurysm may cause pyloric obstruction by pressure; or, by interfering with the coeliac plexus, may give rise to severe vomiting and wasting. Death may occur also from intercurrent maladies, such as heart failure, when there is concomitant valvular disease or muscular impairment. There may, of course, be other manifestations of syphilis in the brain or elsewhere. Pulmonary tuberculosis is not uncommon.

Allusion has been made to dissecting aneurysms. The blood which creeps between the arterial coats might conceivably make its way through the external coverings and cause

death by bleeding, but usually an opening is established back into the lumen through the inner coat. The secondary channel so made may remain open for many years, and at post-mortem examinations the condition has been mistaken for a congenital reduplication of the aorta. The separation of the coats may extend for a great distance, for example from the thoracic aorta to the iliac arteries. In a considerable number of cases the effused blood clots and is ultimately absorbed, a complete cure resulting.

Symptomatology.—The patient's complaint in cases of aneurysm depends very largely on the situation of the tumour. Perhaps the commonest symptom which induces the patient to seek medical advice is *pain*. It is often constant and prominent. The actual distension of the vessel-wall seldom, if ever, causes it, but inflammatory lesions of the great vessels, particularly of the aorta, are sometimes very painful, and the splitting of the intima, already described, may be associated with pain which is sudden and severe. Thus, it is not uncommon for the patient to date his illness from an attack of pain which only lasted a short time. When the artery is situated in the trunk the pain is referred to the surface of the body and is associated with superficial tenderness. Thus, when the first part of the aorta is affected the pain and tenderness, as in heart disease, are distributed over the left side of the thorax, spreading at times down the inner side of the left arm to the elbow or even to the little and ring fingers. With disease of the aorta it is also not uncommon for their distribution to be on the corresponding region of the right side and the right arm. In some cases the pain may spread up into the neck.

As with other tumours, pain is liable to ensue when nerves and other structures are pressed upon, and from this cause aneurysm may be accompanied by continuous pain, which is sometimes of excruciating severity. With the implication of nerves it is of a neuralgic character, and is felt in the peripheral distribution of the affected nerve. From the point of view of sensation, bone is the most important of the other structures, its erosion being usually accompanied by persistent and unbearable aching pain. It is curious, however, that sometimes extensive destruction of bone may take place and yet the patient suffer little or no discomfort, whilst in other cases the pain is so severe that large doses of morphia are required to afford the sufferer any relief.

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In the **limbs**, owing to the absence of important viscera, there may be very few symptoms to call the patient's attention to his disease. Pain, which may be quite absent, is seldom severe, and is generally of an aching character. Sometimes there is an unpleasant sensation of throbbing, or the patient may himself discover the existence of a swelling. In a fair number of instances the aneurysm, unnoticed by its bearer, is accidentally found during a medical examination for some other condition.

Where the **trunk arteries** are implicated the symptoms are naturally much more prominent and varied, but even here there may be a complete absence of complaint, and the aneurysm may be discovered by chance. As the symptoms depend on the situation, it is necessary to consider the different parts of the great vessels in detail. The **lower part of the abdominal aorta and the external iliac arteries** are not in close association with important viscera, and hence arterial tumours arising from them are not likely to give marked symptoms. Pain, however, may be severe from erosion of the spine. An aneurysm of any artery is liable to interfere with the blood supply to parts beyond it. Thus, with abdominal aneurysm the lower limbs may receive an insufficient supply of blood. The arterial flow may be adequate when the limbs are at rest, but during movement more blood may be required than the legs can obtain. If this is the case, after some exertion the muscular power in one or both legs may fail, and the patient may be obliged to stop and remain still until his muscles have recovered. This "intermittent claudication" was first described by Charcot in a case of aneurysm of one of the iliac arteries.

The fact that the intestines are freely movable within the abdominal cavity saves them from injurious compression, but the ureters, being in a more fixed position, may be obstructed and hydronephrosis may result. Aneurysms of the **internal iliac arteries**, which lie in the enclosed box of the pelvis, are liable to compress the rectum and to interfere with the bladder and so give rise to difficulty in defaecation and troubles with micturition. The pelvic portion of the ureters may also be compressed, or considerable pain may be caused by pressure on nerves (e.g. the sciatic), or by erosion of bone.

The commonest situation for an **abdominal aneurysm** is at the origin of the coeliac axis from the aorta, and the tumour, in addition to eroding the spine, may project and press

on the plexus of nerves in front. From this may arise pain and signs of interference with the innervation of the stomach, such as dyspepsia, vomiting, and wasting. The stomach itself, near its pyloric end, may be reached by the swelling, and in consequence there may be hæmorrhage from its mucous membrane, vomiting of blood and mælena; there may even be pyloric obstruction, the whole group of symptoms suggesting either a simple or a malignant ulcer. Rupture into the stomach is very rare, bursting taking place more commonly into the loose retroperitoneal tissues. When the latter occurs death does not necessarily ensue at once; indeed, recovery may follow in rare instances. The main symptoms of such a rupture are collapse, thirst, sighing respiration and increasing anæmia (i.e. signs of internal hæmorrhage), and in addition there may be aching pain in the back. When the pre-existence of an aneurysm is unknown an exact diagnosis may be very difficult of attainment, and laparotomy has been performed several times under the impression that the collapse was due to a perforation of the stomach or bowel.

The **descending thoracic aorta** is less frequently affected than any other portion of the great arteries of the chest. In no position is the disease more obscure, yet in none does the swelling reach a larger size. Symptoms may be entirely absent, but, on the other hand, pain may be more severe with an aneurysm in this situation than in any other, because of erosion of the vertebral column. The severity of the symptoms depends more on the direction of the growth of the tumour than on its size; expansion forwards leading only to some collapse of the base of one or other lung (usually the left), and perhaps a little shortness of breath, while pressure of even a small tumour on the spine leads to bone erosion and often agonizing pain. The patients assume a characteristic attitude to relieve their suffering, sitting up in bed and supporting the head on the arms and knees. Occasionally the aneurysm erodes into the spinal canal and, pressing upon the cord, leads to paraplegia, but this occurs only very rarely. Still more rare is rupture into the spinal canal with its sudden association of collapse and paraplegia. It is with aneurysm of the descending thoracic aorta that the œsophagus is most likely to be implicated, and in consequence there may be difficulty in swallowing, but this is not at all common. Weeping may take place into the

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gullet and give rise either to hæmatemesis or melaena, which may continue for some weeks and then end by a fatal rupture. A few cases are on record where the passage of an œsophageal bougie has led to the fatal perforation of an aneurysmal sac, and, in modern times, no bougie should be passed down the œsophagus for the relief of a supposed stricture until the absence of an aneurysm has been demonstrated by means of X-rays. An aneurysm in this situation most frequently causes death by long-continued suffering, aided, perhaps, by some pulmonary complication such as bronchitis. Sudden death may, of course, follow rupture into the œsophagus, the spinal canal, the pleuræ (usually the left), or the tissues of the posterior mediastinum. Sometimes it leads to complete absorption of the adjacent portions of ribs and a pulsating tumour projects on the posterior surface of the body to the left of the spine; nevertheless, external rupture in this region is excessively rare.

When considering aneurysm of the **transverse portion of the thoracic aorta**, it must be recalled that this portion of the aortic arch is situated in the narrowest part of the thoracic cage and in close proximity to numerous important structures. Hence even small aneurysmal tumours in this situation are almost certain to exert pressure on one or more of these and, by doing so, to cause symptoms. The objective signs may be few or absent, but the symptoms are more prominent than are those produced by an aneurysm in any other situation. Pain may arise either from erosion of the sternum in front or of the spine behind; and neuralgia down the left arm and over the left side of the neck may follow pressure on the cervical and upper thoracic nerves. A more constant symptom is dyspnœa, which may be due to a variety of causes. The lung may be displaced or partially collapsed, or there may be pressure upon the trachea or one of the bronchi, usually the left. In cases where pressure is exerted upon the respiratory apparatus there is frequently, in addition to dyspnœa, some hæmoptysis due either to congestion of the lung or of the mucous membrane of the respiratory tract, or to weeping of the aneurysm into the trachea or bronchus. Rupture, particularly into the left bronchus, is no uncommon termination. Constriction of the larger air-tubes causes stridor, especially on exertion. Compression of a bronchus may lead to collapse of the lung, or bronchiectasis and fibrosis of the lung may ensue as the result of

the retention and putrefaction of bronchial secretion. In these circumstances the wasting, hectic temperature, and cough may lead one to suspect pulmonary tuberculosis. Any implication of the respiratory apparatus usually causes an irritative cough. Any of the nerves traversing the thorax may be pressed upon. The least likely to escape is the left recurrent laryngeal, since it winds round the aortic arch. Its destruction produces paralysis of the left vocal cord, causing the cough to become brassy in character and the voice hoarse and toneless. The vagus itself is much less often affected, but, should this occur, tachycardia, dyspepsia, and vomiting may result. Irritation of the vagus may lead to a reflex spasm of the larynx which may prove fatal. Unilateral paralysis of the diaphragm may be produced by pressure on one of the phrenics, but is not common. In some cases the cervical sympathetic is involved, its stimulation causing flushing and sweating of one side of the face and dilatation of one pupil, its destruction the reverse. Inequality of the pupils is common in thoracic aneurysm, but is usually due to interference with the blood supply rather than to affection of the sympathetic. Other structures which may be pressed upon are the œsophagus and the thoracic duct, but they usually escape. Blood-vessels may be implicated in two ways. They may be involved in the arterial disease, or compressed by the tumour from without. In the case of the arteries the former is the more common. The pupil may be dilated when disease narrows the entrance to the carotid, and the size of the radial pulse may be reduced when the subclavian is affected. The veins are more likely to be compressed. This is especially true of the left innominate, so that congestion and swelling of the left side of the head and of the left arm are not uncommon symptoms; occasionally this has been associated with clubbing of the finger-tips of the same side. In spite of the proximity of so many important structures, both physical signs and symptoms are sometimes very inconspicuous.

Death from hæmoptysis is most common in aneurysm of the transverse arch, owing to its liability to rupture into the left bronchus. Rupture may also take place into the trachea, the left pleural cavity, the mediastinum, or the left innominate vein. Death is usually instantaneous with rupture into the pleural cavity, and follows after a few days of preliminary weeping when the opening is into the air-passages; but rupture into the innominate

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vein is not necessarily followed by death, an aneurysmal varix being formed which gives rise to its characteristic signs.

In contrast to the transverse portion of the arch, **aneurysm of the ascending aorta** usually affords obvious physical signs, but frequently very little in the way of symptoms. Pain is one of the most common, being either constant or paroxysmal and spreading down one or other arm. It is often of the character of angina pectoris, and is generally associated with superficial tenderness in the area of its distribution. It is also liable to be caused by erosion of the ribs or sternum when the aneurysm bulges forwards. Cough and dyspnoea are not so frequent as when the transverse arch is involved, but the right bronchus is sometimes pressed upon. The disease of the ascending aorta from which the aneurysmal dilatation arises is liable to invade that portion which is nearest to the heart, and so to affect the aortic valves and possibly the coronary arteries. Hence obstruction of the orifice or incompetence of the aortic valves is not an infrequent complication, and a fair number of cases are accompanied by symptoms of heart failure. Pressure on the superior vena cava may cause swelling of the face and of both arms, and later an enlargement of the veins in the abdominal wall, as the result of an attempt to return the blood from the upper portion of the body by way of the iliac veins and the inferior vena cava. In some cases the innominate artery is involved in the aneurysm, while in others its orifice may be partially occluded by disease of the arterial walls; either of these circumstances is likely to lead to diminution of the right radial pulse and dilatation of the right pupil. The patient himself may notice the development of a throbbing tumour projecting in the front of the right chest.

An aneurysm of the first part of the aorta may rupture into the right bronchus, the right pleural cavity, the anterior mediastinum, or on the external surface, though the last is rare. Communication may be established between the aneurysmal sac and the superior vena cava (producing an aneurysmal varix), the pulmonary artery, or one of the auricles. Aneurysm of that portion of the artery which is covered by the pericardium has been placed in a class by itself because of the frequency with which it leads to sudden death by rupture into the pericardial sac without previous symptoms. This occurs in spite of the fact that in such cases the aortic valves are usually incompetent.

Death from rupture into the pericardium is usually instantaneous but, in rare cases, may be delayed for a day or two, or even for five days, as in a case admitted into hospital under the care of my late colleague, Dr. Murrell.

Aneurysms of the **great branches of the aorta** in the neck and upper limbs form obvious tumours, but do not usually implicate important structures, so that the patient only complains of the swelling and a sensation of throbbing. In the case of the innominate artery, most of the tumour is situated inside the thorax, and may cause some erosion of the right first rib and of the sternum, besides displacing the clavicle. The right recurrent laryngeal nerve hooks round the right subclavian artery, dilatation of which may compress the nerve and lead to paralysis of the right vocal cord with its attendant hoarseness and alteration in voice. Axillary aneurysm may cause pain by pressure on the nerves to the upper limbs, or swelling of the arms from obstruction of the veins.

Physical signs.—In the examination of a patient the chief sign of aneurysm is the presence of a tumour which pulsates, this pulsation being found to be expansile when the tumour is grasped in the fingers. Pulsation may be communicated by a large artery to a tumour lying over it, but this pulsation is not expansile, and on lifting the tumour away from the vessel the pulsation ceases. If the artery be compressed between the heart and the aneurysm the pulsation will diminish or cease, according to the degree of compression. On listening over the tumour a systolic murmur can often be heard, but care must be taken not to compress the vessel, for this in itself is sufficient to produce a murmur. In the case of abdominal aneurysms a bruit may be heard on listening over the spine at the back. Still, a murmur is not present in all cases, and the absence of a murmur does not necessarily mean that there is no aneurysm.

Aneurysms situated in the enclosed box of the thorax only become obvious when they project through the thoracic walls on to the surface. Sometimes large external swellings, with the typical expansile pulsation, are produced, especially when the aneurysm arises from the ascending aorta. In many cases, however, the swelling is very small, or there may be no external projection at all; but, even in these, pulsation can sometimes be seen or felt in abnormal situations, such as on one or other side of the upper part of the sternum, or in the left interscapular region behind. A

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thrill may be detected on palpation, and also a peculiar and characteristic shock during cardiac diastole. In the visual examination of a case of suspected aneurysm the patient should be placed in a good light, and the front of the chest inspected by viewing it from side to side as well as directly.

On auscultating the chest, besides the systolic murmur a diastolic murmur may be heard, but this only occurs when the aortic valves are incompetent, and is not, of itself, a sign of aneurysm. Of more importance is a peculiar ringing character of the second sound, which is audible whenever the aorta is dilated in the vicinity of the heart. Fusiform dilatation of the aorta, besides being accompanied by a systolic bruit over the upper part of the sternum, causes the arch to ascend, so that it can be felt pulsating above the sternal notch, and the subclavian arteries become situated abnormally high in the neck. The diminution of one or other radial pulse and the dilatation or contraction of one or other pupil have been already alluded to. A decided difference in the blood-pressure between the two arms may be found. When the aneurysm touches the trachea or large bronchi its pulsation causes a movement of the trachea as a whole. This is known as the "tracheal tug," and can be felt by pressing up the cricoid cartilage by a finger on each side. The trachea is tugged down with each pulsation of the aneurysm.

Most of the other physical signs of aneurysm in the thorax are merely those of a tumour, but it must be remembered that aneurysm is the commonest form of intrathoracic tumour, especially in middle life. A tumour in the thorax always means more or less displacement of other structures, and it may be possible to demonstrate this displacement by physical examination. The lungs are almost certain to be compressed and displaced to some extent, and if the tumour comes at all near the external wall of the thorax the percussion resonance will be diminished or abolished and the breath sounds will become feeble. Such signs are most likely to be found to the right of the sternum when the ascending aorta is at fault, and in the first left interspace when the transverse arch is concerned. Similar signs may be found at the back, on the left side of the spine, with aneurysm of the descending thoracic aorta. With mere dilatation of the aorta there will be dullness or impaired resonance over the sternum, tending to spread a little to the right side. The heart is liable to be displaced when

the aneurysm is near it; usually the base of the heart is pushed downwards so that the organ comes to lie more transversely, as is well shown in skiagrams. It is important to distinguish between displacement of the heart and enlargement from dilatation and hypertrophy, for enlargement of the heart is no uncommon accompaniment of aortic aneurysm. It is not due to the aneurysm, but is the result of heightened blood-pressure, incompetence of the aortic valve, or muscular impairment.

Apart from displacement, pressure on intrathoracic structures may lead to other changes which would be noticeable to the observer. For instance, pressure on the great veins causes congestion and swelling of the head and upper limbs, either bilateral or unilateral in distribution, and sometimes leads to the establishment of an obvious collateral circulation by anastomoses between the superficial abdominal veins, and the iliac veins and the inferior vena cava. In such cases the flow of blood in the abdominal veins would be downwards. Pressure on the trachea or bronchi causes stridor, which is at once audible on approaching the patient. Pressure on one bronchus diminishes the air entry on that side, and hence lessens the respiratory murmur. There may even be complete collapse of the lung on the affected side, with consequent impaired percussion note and absence of movement and breath sounds. In other cases the lung is overdistended because the air cannot escape, and the poor movement with hyper-resonant percussion note and diminished breath sounds may suggest the diagnosis of pneumothorax. The signs may be partial instead of general if a branch is compressed instead of the main bronchus. The retention of secretion in the bronchi may lead to dilatations in which the secretion becomes septic. The physical signs of this bronchiectasis with its surrounding fibrosis of the pulmonary tissue may simulate those of an advanced tuberculosis, especially as there is usually a hectic temperature. Even pressure on the lung tissue itself may lead to a local congestion and hæmoptysis, and over the involved area râles may be heard suggesting an early tuberculous infiltration. The difficulty of diagnosis is increased because pulmonary tuberculosis is sometimes found accompanying aneurysm. Compression of the air-passages is further very prone to set up a bronchitis, the physical signs of which would be found.

Pressure on nerves may also cause ascertainable signs. The left recurrent laryngeal is the

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most frequently concerned, and may gradually have all its fibres destroyed. Those which supply the abductors of the vocal cords are first affected. Their paralysis does not alter the voice, so that before any change of voice is noticed a laryngeal examination may reveal that the left vocal cord is immobile in the mid-line in the position of phonation. Later, with the destruction of the other fibres, the cord becomes completely paralysed, first losing its tension and then lying in the cadaveric position, half-way between adduction and abduction, and flapping idly in the air-stream. The voice then becomes hoarse and toneless, and may be reduced to a whisper. The right vocal cord may be similarly paralysed by involvement of the right recurrent laryngeal by an aneurysm of the right subclavian. Loss of movement on one side of the diaphragm follows the destruction of a phrenic nerve, the left being the more frequently affected, but the lesion is very rare. The signs of implication of the sympathetic have already been dealt with.

It is uncommon for destruction of the vertebrae to go so far as to produce definite physical signs, but kyphosis from falling in of the vertebral bodies has been known, and sometimes pressure on the spinal cord produces spastic paralysis of the lower limbs.

The discovery of the Röntgen rays has provided a certain method of ascertaining the size and position of thoracic aneurysms. The pulsating shadows can be seen on the screen and the cardiac displacement observed; or photographs can be taken and so alterations in the size of the tumour recorded during the progress of the disease. Small dilatations in the transverse portion of the arch may be missed in an X-ray examination, but elsewhere it is possible to be certain of the presence or absence of an aneurysm and of its exact position in the chest. With abdominal aneurysms the results are much less satisfactory, and the fluorescent screen is not of much assistance in forming a diagnosis.

Diagnosis.—An aneurysm situated in a limb can rarely be confused with anything else. A tumour connected with an artery which shows the typical expansile pulsation must be an aneurysm. When, however, the sac has become filled with clot the pulsation disappears, and such a solid tumour may be mistaken for a sarcoma. Hence the possibility of a healed aneurysm in a man in early middle age with a history of past syphilis or a positive Wassermann reaction must be remembered.

In the abdomen, also, the typical expansile pulsation is unmistakable. Sometimes a tumour situated over the aorta has pulsation communicated to it, but this pulsation is not expansile, and the tumour can often be lifted away from the aorta, when the pulsation ceases. Undue throbbing of the normal aorta sometimes leads to confusion. This is considered under AORTA, DYNAMIC PULSATION OF. In aneurysm, a murmur can usually be heard over the tumour, but a similar systolic murmur can easily be obtained by pressing the stethoscope over the normal aorta. A murmur heard over the spine at the back is more valuable if present, but is by no means always found. Rupture of an abdominal aneurysm into the retroperitoneal tissues may be difficult to diagnose if the previous existence of an aneurysm is unknown. The signs are those of internal hæmorrhage associated with pain in the back, but several of the cases have been mistaken for perforation of some part of the gut.

In the thorax, diagnosis is simple when there is an expansile tumour projecting on to the surface, but without this a careful consideration of the symptoms and a long and detailed examination is required. As already mentioned, aneurysm is the commonest form of intrathoracic tumour in men in early middle life, and its presence is probable when there are signs of pressure within the chest. This probability is increased if the Wassermann reaction is found to be positive. In any suspicious case it is important to compare carefully the pulses and pupils, to examine the larynx even when there is no alteration of voice, and to test for tracheal tugging. Aneurysm almost invariably affects only one vocal cord, so that a bilateral lesion would suggest some other form of tumour. Aneurysms are more prone than malignant growths to erode opposing bone. The pulsation of an aneurysm is forcible, like that of the apex beat of the heart, and, with the diastole, a very characteristic shock is communicated to the palpating hand. With pulsating solid tumours these characters are absent.

Sometimes *empyemata*, when near the heart, and also pointing on the body surface, pulsate, but this pulsation is diffuse and has none of the characteristics of aneurysmal pulsation. Still, every doubtful case should be submitted to an X-ray examination, which will nearly always definitely decide whether an aneurysm is present or not.

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The thoracic aorta, like the abdominal, may at times pulsate so excessively as to give rise to the suspicion that an aneurysm is present, especially when it is associated with regurgitation through the aortic valve. This pulsation will be felt to the right of the sternum and above the sternal notch, but the fluorescent screen should at once eliminate any possibility of mistake from this cause.

Prognosis.—This depends very largely on the situation of the tumour. In the limbs it may be efficiently treated, if the vessels are otherwise healthy; whereas in the trunk it is much less accessible, and treatment is proportionately unsatisfactory. It is to be remembered that spontaneous cure by thrombosis may take place in any aneurysm, but is more probable in those occurring in the limbs. As a rule, a patient suffering from thoracic aneurysm does not live for more than two to three years after the disease is first diagnosed; but occasionally, even without clotting in the sac, he may live ten or more years under observation. The progress of a case under treatment affords help in estimating the future chances, but too much stress must not be laid on the relief of symptoms, since discomfort can often rapidly be relieved by appropriate treatment without apparently any real delay in the steady progress to a fatal issue.

Treatment.—The treatment of aneurysm in the limbs is mainly a matter for the surgeon. The general lines adopted are either to induce clotting in the sac by limiting or abolishing the blood-flow through it, or to extirpate the sac completely by the method originally devised in the second century by Antyllus. The blood-flow through the sac may be controlled either by compressing or tying the artery between the aneurysm and the heart, or, if this operation is impracticable, by tying the artery or arteries on the distal side of the sac. Where possible, the operation of Antyllus is now preferred by surgeons, provided the vessels generally are fairly healthy; and the results are usually excellent. If the blood-vessels are extensively diseased there is some danger of gangrene of the limb supervening from the failure of the establishment of a collateral circulation. Surgical measures have even been devised for the treatment of aneurysms of the trunk. For instance, the distal arteries may be ligatured for an aneurysm of the innominate, subclavian, or carotid, and the abdominal aorta has been successfully compressed by means of a special tourniquet, although the

procedure is decidedly dangerous. Again, attempts have been made, with some success, to encourage the formation of thrombus in the sac by introducing metallic wire into it and by scarifying the internal surface of the cavity by means of needles. However, cases suitable for these heroic procedures are rare, and the treatment of trunk aneurysms usually depends upon measures devised by physicians.

The general aim and object of treatment has been to cure the aneurysm by bringing about thrombosis, following Nature's method. It should be recognized that this is not free from danger, since the main vessel may also be thrombosed, or a portion of the newly formed clot may become detached and give rise to embolism elsewhere. In order to increase the coagulability of the blood, various drugs have been employed, of which gelatin and calcium lactate may be mentioned. The subcutaneous injection of gelatin (200 c.c. of a 2-per-cent. solution with 7 per cent. of sodium chloride) has been advocated by Lancereaux, who gives up to forty injections at intervals of a week. The favourable results recorded by the introducer have not been confirmed by other observers, and at least one fatal accident has happened because the gelatin solution used has not been sterile. The exhibition of lactate of calcium has not, so far, been found to be followed by any definite formation of clot in the aneurysmal sac.

Another method aims at the same result by trying to diminish the general blood-flow and so lessen that passing through the sac. Complete rest very materially reduces the rate and force of the heart, and the arterial blood-pressure may also be diminished by a very restricted diet. The further reduction of the blood-pressure by repeated abstractions of blood has been advocated by some. The elaboration of this method was due to Tufnell, and since his time it has been very widely employed. The patient is kept at absolute rest for a period of many months, and his intake, both solid and liquid, is reduced to the lowest possible point. The diet recommended by Tufnell is as follows:

Breakfast: 2 oz. of white bread and butter, 2 oz. of cocoa or milk.

Dinner: 3 oz. of broiled or boiled meat, 3 oz. of potatoes or bread, 4 oz. of water or light claret.

Supper: 2 oz. of bread and butter, 2 oz. of milk or tea.

The total intake on this diet is ten ounces of

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solid and eight ounces of fluid in the twenty-four hours. It is recommended that this regimen should be maintained in all its strictness for a couple of months, after which the diet may be increased somewhat, but the complete rest must be continued for several more months. The small amount of the intake generally results in constipation, which requires correction by the use of aperients or enemata, but except for this it is advised that no drugs be administered to the patient. Under this régime the symptoms of which the patient complains undoubtedly get less or disappear, but it is very doubtful whether any larger proportion of aneurysms diminish in size or coagulate than under a less uncomfortable method. Certainly in several cases where this treatment has been carried out with great strictness for many months the aneurysm, post mortem, has been found to have no trace of clot in it.

These discouraging results have led many physicians, at the present time, to direct their treatment rather to the relief of symptoms than to any attempt to cure the disease. It is obvious, too, that the number of patients who are suitable for the Tufnell treatment, and at the same time willing to undergo it, must be very limited. It has been found that the distressing symptoms, pain and dyspnoea, may often be completely or partially relieved by methods which involve much less time and discomfort, and that, after relief, a patient may with care live and even follow a light occupation with a fair amount of comfort, while the prospects of cure by thrombosis, though slight, are as good as with other methods. The pain and dyspnoea usually diminish after a few days' rest in bed, and after a few weeks often disappear, particularly if, at the same time, there is some limitation of the diet and if iodide of potassium be given in moderate doses (5-10 gr.) three times a day. The iodide will often relieve, especially in syphilitic cases, even when the patient is up and about, though the exact method by which it brings about amelioration is unknown. Iodides are supposed to lower the blood-pressure, but their influence in this direction is very difficult to demonstrate by the sphygmomanometer, and any reduction of the blood-pressure does not give a satisfactory explanation of the relief afforded.

Individual discomforts may require treatment. Under the regimen outlined above all the symptoms complained of are usually relieved very considerably. It may, however, take several days before the improvement

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manifests itself and, in the meanwhile, special treatment may be necessary. This will also be required in the cases in which relief is not afforded by more general measures. Of these symptoms the most important is pain, which may be of excruciating severity. For this it is necessary to give morphia in sufficient amount, and in bad cases it may have to be gradually increased to a considerable extent. It is best for the physician to administer the drug himself by hypodermic injection. Cough, which is a very constant symptom of aneurysm, may demand some linctus containing morphia or heroin. Dyspnoea is generally best relieved by rest, but is liable to be increased by intercurrent pulmonary affections, such as bronchitis, bronchiectasis, and tuberculosis. In these circumstances some assistance may be obtained by the exhibition of expectorants such as ammonia; and if there is decomposition of the sputa in dilated bronchi, carbolic acid or creosote in minim doses may be used as disinfectants, or antiseptic inhalations may be employed. It is important to remember that in a large number of cases the use of these drugs need be only temporary, and that they may be gradually diminished as the symptoms decrease in intensity.

After the relief of symptoms the patient should still be kept in bed for several weeks. He may then be allowed very gradually to get up, the effect of each slight extra exertion being noted. If there be a return of pain or other distressing symptom he should again be put back to bed for some weeks. When the patient has satisfactorily passed through a prolonged convalescence, he may be allowed to undertake some light occupation, but must be warned against any severe exertion. He should be examined periodically to make sure that the aneurysm is not increasing in size, and it is generally advisable to continue the administration of the iodide. A. M. GOSSAGE.

ANEURYSM, ARTERIO-VEINOUS, is the result of a direct communication between an artery and a vein. It is seen most commonly in the limbs as a sequel of stabs or gunshot wounds, and at one time was comparatively common at the bend of the elbow from injury inflicted on the brachial artery in the process of venesection. Sometimes it is found in the chest or abdomen from the bursting of an aneurysm into an adjacent vein. The artery may open directly into the vein, when the condition is known as *aneurysmal varix*; or the

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blood may have been effused into the tissues before passing into the vein. In the latter case a sac wall is formed from the tissues, and the tumour is called a *varicose aneurysm*.

Symptomatology.—In aneurysmal varix there are great distension of the vein and, naturally, a blocking of the blood-flow into it from the periphery. Consequently, in the limbs, there are venous engorgement, possibly oedema, and, with lapse of time, an increased growth of the limb and its hair. When situated in the thorax there are cyanosis and swelling of the face and upper limbs of sudden origin, while swelling and oedema of the lower limbs occur in the rare cases in which the aneurysm is in the abdomen. Similar but less pronounced phenomena accompany varicose aneurysm. On examination either a greatly distended and pulsating vein will be found or a pulsating tumour at the side of a more moderately distended vein; and, on auscultation, a characteristic continuous murmur (which has been likened to a fly buzzing in a box) will be heard, the sound being accentuated with each pulse. If this condition occurs in the trunk, the distended vein will be hidden, but the peculiar murmur will be heard.

There is no special **treatment** applicable to cases in which the abnormal communication is established between vessels of the trunk. In the limbs the swelling may be compressed by an elastic support, or the artery may be ligatured above and below the lesion. Ligature is practically always necessary in varicose aneurysm owing to the risk of rupture and escape of blood.

A. M. GOSSAGE.

ANEURYSM, CIRROID (*syn.* Arterial Angioma).—A congeries of vessels composed of enlarged branches of an arterial trunk. There is probably some antecedent congenital anomaly, though the advent of the swelling is often preceded by an injury. The scalp is the most usual situation, the temporal artery being that most commonly affected. The condition is chiefly met with in adolescents and young adults. Beginning in the smaller branches, an arterial overgrowth occurs, which gradually spreads until perhaps the main trunk is involved, or the anastomosing branches of neighbouring vessels. To the general enlargement are added tortuosity and zones of constriction and dilatation until a pulsating tumour is formed, composed of tortuous and enlarged arteries and feeling like a mass of hard worms. In the case of the temporal

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artery the swelling may spread until practically the whole scalp becomes affected; gutters may be produced in the bone by the overlying vessels. If the ophthalmic artery takes part or is the site of the lesion, pulsating exophthalmos (*see* ORBIT, AFFECTIONS OF) may result. The most serious symptom of a cirroid aneurysm affecting an artery supplying the head is a loud systolic murmur, audible to the patient and often causing him great discomfort and loss of sleep. Neither rupture nor spontaneous cure is to be anticipated, though profuse and dangerous hæmorrhage may follow wounding of the area. Occasionally extensive ulceration occurs.

Treatment.—When the murmur is troublesome or the condition is spreading, excision, allowing a wide margin, may be undertaken in suitable cases, the bleeding being controlled as far as possible by an elastic tourniquet around the head, by previous ligature of the supplying trunk, or by injecting into it boiling water. When the lesion is too extensive for excision or unsuitably placed, amelioration may be attempted by proximal ligature alone. An alternative method, when the aneurysm is accessible, is electrolysis.

FREDERICK LANGMEAD.

ANEURYSMAL VARIX (*see* ANEURYSM, ARTERIO-VEINOS).

ANGINA CRURIS (*see* ARTERIAL DEGENERATION).

ANGINA, LUDWIG'S (*see* LUDWIG'S ANGINA).

ANGINA PECTORIS.—The term angina pectoris is applied to the more severe grades of cardiac pain. *Pseudo-angina* is an unsatisfactory term which is applied to cardiac pain of an anginal type occurring in patients in whom there is no evidence of organic cardio-vascular disease. In some of these patients the neurotic element is more in evidence, in others the toxic. It should be emphasized that there are all grades of severity of cardiac pain, and that there is no sharp and clear line of demarcation between the mildest pseudo-angina and the angina pectoris gravior of Heberden. Anginal pain is evidence of exhaustion of the heart-muscle.

Etiology.—Angina is essentially a symptom of advancing years. It is rare before forty. As a man ages his arteries lose their elasticity, the arterial walls thicken and their lumina diminish, with the result that the blood

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supply to the various organs is lessened and their functioning power impaired. In the case of the heart, this is shown by a diminution of its reserve force and the ease with which shortness of breath and cardiac pain are induced—both signs of cardiac exhaustion.

Cardiac pain is common in women, but the anginal type of pain more frequently attacks men. The active man of business, the "hustler" endowed with boundless energy, who works year in and year out at top speed and is incidentally a high feeder, is a frequent victim.

High blood-pressure is an outstanding feature in one type of case.

Syphilitic aortitis is recognized as a cause of very severe cardiac pain, and it is noteworthy that cardiac pain of the anginal type is much commoner when the disease affects the aortic valves than when the mitral valve alone is involved.

Predisposing causes.—Since anginal pain is the result of stimuli passing up from the heart and impinging on the nervous system, it is clear that the degree of pain will depend on the relationship of these two factors.

Freedom from pain will depend in many men on the careful preservation of the balance between the power of the heart for work and the sensitiveness of the nervous system.

Myocardial disease, coronary sclerosis, anaemia, febrile illnesses, toxæmia, underfeeding, and any other cause impairing the nutrition of the myocardium will render it more readily exhausted. A much less degree of effort and strain will then induce pain and cardiac distress.

On the other hand, any factor which increases the sensitiveness of the nervous system will also increase the liability to pain.

An exhausted nervous system is an irritable nervous system, and such is very often the outcome of prolonged business worries, overwork, anxieties, or recurrent insomnia.

Exciting causes.—The immediate cause of an attack is generally some extra physical effort, such as hurrying for a train or struggling against a wind. Effort immediately after a meal is more likely to induce the pain, and many patients have noticed that sudden exposure to cold brings on præcordial pain, anginal in type.

Emotion is a well-recognized factor; the reason is not obvious, but the increased heart-action and sudden variation in blood-pressure may be the explanation.

The onset of fibrillation of the auricle (delirium cordis) or an attack of paroxysmal tachycardia occurring in a heart with diminished

reserve, by greatly increasing the rate of the heart and shortening diastole, may cause rapid exhaustion of the heart-muscle and the appearance of severe cardiac pain, which passes away when the normal rhythm is restored. Rapid heart-action of this nature is not a response to stimuli acting on the pacemaker of the heart through the nervous system and compensatory in character; it is rather a sudden increase in rate independent of the nervous system, imposed on the heart irrespectively of the condition of the remainder of the vaso-motor system, and therefore much more liable to cause sensory distress.

Description of an attack.—The onset may be sudden and unexpected, or the development of the symptoms may be a gradual crescendo.

The pain when fully developed is intense and agonizing, and described as tearing, gripping, gnawing, and unbearable, or even beyond description. It is situated either in the middle line or a little to the left, about the level of the 3rd, 4th, and 5th intercostal spaces, and may "go right through to the back" or spread round the chest-wall towards the left scapula. When the initial pain is situated above the level of the nipples it is more likely to extend into the arms. When it is most intense at or below the nipple level it frequently passes round and through to the back. (Fig. 5.) It may become so severe that the patient remains motionless, afraid even to breathe; in other cases, however, there is a peculiar restlessness, and even a desire to move about during the paroxysm.

Together with the pain there is usually a sense of constriction and pressure, the chest feels as if gripped in a vice and crushed, breathing becomes difficult, and the patient feels suffocated.

The pain may remain localized to the region of the præcordium where it was first perceived, but frequently it spreads to the left shoulder and down the inner side of the arm, sometimes stopping at the inner side of the elbow, but, if the attack is prolonged and severe, extending to the wrist or fingers. Finally, both arms may be involved. Often a sensation of numbness and tingling precedes the pain in the arm.

Accompanying the pain, the power of the hand and arm may temporarily be impaired.

Pain is occasionally experienced in the left side of the neck, the left jaw, and the occipital region.

The above is the usual sequence of events, but at times the first indication of an attack is pain or tingling above the inner side of the left

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elbow, or more widely spread in the left arm, and this pain spreads from the arm to the chest.

Cardiac pain is nearly always left-sided, but sometimes without apparent reason it begins to the right of the middle line and spreads thence into the right shoulder and down the right arm, the character and intensity of the

apprehension. The dread of imminent death dominates the mind and is expressed in the countenance.

The pulse may remain little altered during a paroxysm, and there is no constant finding. It may become extremely feeble and irregular, or, on the other hand, in some patients the pressure is found to be raised. Wheezings in the chest, both inspiratory and expiratory, are sometimes present, accompanied by a sense of suffocation; this wheezing may persist after the anguish has subsided. Vomiting rarely occurs, but eructation of wind is frequent, and is often due to air-swallowing. Hiccough or persistent yawning is at times a troublesome symptom.

With the termination of the attack large quantities of pale urine may be voided. Although the relief from the excruciating pain may be complete, the patient is usually very limp and exhausted and disinclined for effort, and a dull aching over the præcordial area may persist for hours.

Tenderness of the chest-wall is often noted during an attack, and may persist. The left breast is tender when handled, and the pectorals also are sensitive to pressure. Pinching the left sterno-mastoid and trapezius will sometimes give rise to pain, in fact may act as the exciting cause of a paroxysm. More rarely the skin itself is hyperalgesic—a feature which is more commonly observed in women.

Although the paroxysm may begin suddenly and without any warning, there is usually a short period of discomfort, frequently described by the patient as "indigestion." This is a feeling of pressure or fullness about the middle and lower third of the sternum, and is often ascribed to flatulent distension. If this has appeared as the result of effort, cessation of the effort usually suffices to cause its disappearance. If, however, the significance of the "indigestion" is not appreciated and the effort is continued, the discomfort increases and merges into a disabling pain. This transition may be sudden and the pain agonizing.

The distribution and the manner of spread of the pain in these cases are an excellent example of the referred pain described by Dr. Head and Sir James Mackenzie (Fig. 5). The segments chiefly involved are the lower cervical and upper dorsal. The first to be affected are the 3rd, 4th, and 5th dorsal, but the 1st and 2nd dorsal are also soon involved and account for the pain felt in the inner side of the arm. Then the lower cervical segments are

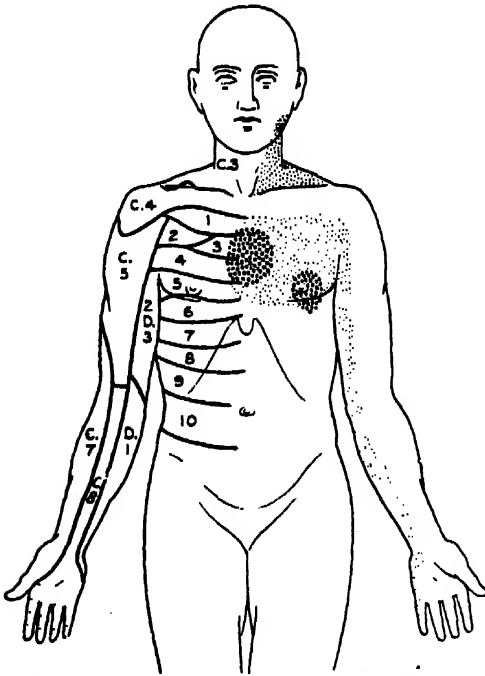


Fig. 5.—To show the common distribution of pain in angina pectoris (a) above the level of the nipples, with a tendency to spread down the arms; (b) at and below the nipples, with a tendency to extend through to the back. The segmental distribution of the cervical and dorsal nerves is indicated.

pain and the associated phenomena being similar to those noted when the pain follows the usual course. This deviation does not appear to modify either the course of the attack or the prognosis.

The duration of a severe attack is generally short and may terminate suddenly; but sometimes the pain waxes and wanes, one paroxysm is followed by another, and the patient, becoming more and more collapsed, sinks into unconsciousness.

With the onset of the pain the face becomes ashen and sunken, a cold sweat breaks out, and the expression is one of extreme anxiety and

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reached and the pain spreads into the fingers. The pain felt in the centre of the back and in the interscapular areas is a further manifestation of involvement of the 5th and 6th dorsal segments.

In rare cases the pain is first felt in the 5th, 6th, 7th, and 8th dorsal segments; it may remain localized to this area, chiefly to the left of the mid-line, or it may gradually spread upwards and finally extend down the arm.

The type of pain just described is much less common in women, but since women also suffer from arterio-sclerosis, chronic Bright's disease, valvular disease, aortic disease, and myocardial degeneration, though less frequently than men, they too sometimes present the typical manifestations of severe angina pectoris. Cardiac pain of a less ominous type, however, is common in women and occurs at a relatively earlier age.

The nervous element bulks large in women, whose nervous system is more easily disturbed by worries, loss of sleep, anxiety, and prolonged mental or physical strain. It consequently responds in terms of pain to a moderate degree of cardiac exhaustion or temporary stress. The pain is often stabbing, or it may be a continuous ache, and it is increased by slight effort or any sudden emotional disturbance. Palpitation is a common accompaniment of this pain, together with a feeling of faintness and exhaustion and a tendency to syncope. The sensation of constriction of the chest as if it were crushed in a vice, and the feeling of extreme sternal pressure so characteristic of the graver type of angina, are rarely met with. The pain in the arm and shoulder is common, but is less clearly limited to the inner side of the limb; rather the whole arm is involved, and the pain is described by the patient as that of neuritis or as rheumatism.

In women, too, it is characteristic to find a much greater degree of hyperalgesia of the skin and a wider distribution of the pain.

At the climacteric there is increased liability to cardiac pain, especially if there is coincident high blood-pressure.

Toxins such as tobacco, tea, and coffee, and also the toxins due to influenza, septic throats, chronic sepsis, and abnormal intestinal conditions, are, probably more frequently than we wot of, the cause of severe cardiac pain approximating in type to that described as common in women. This point of view has been emphasized by Mackenzie.

Prognosis.—A satisfactory prognosis can be arrived at only after full consideration of the

etiology and a clear conception of the underlying pathological changes. A correct prognosis is admittedly difficult, but the outlook in many instances is more favourable than is supposed. The well-known fact that patients may die in their first or second attack, and that the mode of their exitus is both distressing and dramatic, has surrounded the term *angina pectoris* with gloom. Experience shows that most patients live for years after the first twinges.

The severity of the initial paroxysms is no guide to the future. An alarming degree of anginal pain may disappear under rest, and if the mode of life is carefully regulated it may be many years before there is any return of symptoms. On the other hand, a moderate degree of angina may be the forerunner of a steadily progressive pathological condition which is quite untouched by treatment.

The younger the patient the better the prognosis, but severe angina attacking a man in early middle life, in whom there are clinical grounds for a diagnosis of syphilitic disease of the aorta, would certainly justify a serious view in regard to the future.

All signs which point to serious organic disease of the heart or arteries increase the gravity of the outlook. When anginal pain is accompanied or preceded by easily induced shortness of breath, bouts of cardiac asthma, Cheyne-Stokes breathing, alternation of the pulse, and high blood-pressure, it is only corroborative evidence of the proximity of a fatal issue.

The most practical test is the extent to which the patient responds to treatment. When the causes, both predisposing and exciting, responsible for the attacks are definite and can be remedied, there is a fair probability that life will be prolonged for years.

The outlook is also favourable when there is clear evidence of an exhausted and over-sensitive nervous system. With the restoration of the nervous system to its normal balance the liability to cardiac pain disappears. This is particularly the case in women.

It is satisfactory when one finds considerable variation in the degree of effort necessary to bring on cardiac pain, the inference being that there is no permanent irremediable disease of the myocardium and coronary arteries; but if pain is more and more easily induced in spite of rest and care, and if the attacks become more frequent and more severe, it is clear that the

ANGINA PECTORIS

myocardium is growing steadily less efficient, and death may occur at any time.

Treatment.—During the paroxysm the urgent need is relief of the pain and the accompanying shock.

A capsule of amyl nitrite should be crushed and the vapour inhaled. The relief is sometimes immediate, though unfortunately often transient, in which case the inhalation must be repeated. Amyl nitrite acts as a rapid vaso-dilator and lowers the blood-pressure. It can be administered by the mouth—for example, one or two drops may be dissolved in spirits of chloroform and sipped in a draught of water.

Unfortunately amyl nitrite often fails, and other means must be employed.

Alcohol is also a vaso-dilator, and a hot drink of brandy-and-water frequently gives speedy relief.

One teaspoonful of the following mixture in several ounces of water is valuable as a strong carminative, and generally relieves the sensation of flatulence.

Ry	Menthol, gr. v.	
	Sp. ammon. aromat.	} āā ʒi.
	Sp. chlorof.	

In the severer cases morphia should be given hypodermically and speedily, either alone or with atropine. One-sixth to a quarter of a grain should be given to begin with, and followed by further injections as required. It is said to be less likely to cause nausea when combined with atropine. The relief from the terrible pain will lessen the degree of shock experienced by the patient, and thus tend to diminish and prevent the onset of collapse. The presence of chronic Bright's disease is no justification for withholding morphia.

I have never seen any benefit from hypodermic injections of strychnine during the paroxysm. The pain can, of course, be dulled by a whiff or two of chloroform. Relief is also experienced from the free administration of oxygen; it should be continued for an hour or more after the paroxysm if there is great exhaustion.

For some days after an attack complete rest is advisable. Then, after carefully investigating the causes that have led up to the paroxysm and forming an opinion as to the degree of organic disease and the temperament of the patient, he must be carefully instructed as to his mode of life.

It is, I think, well to explain quite frankly

that the pain is evidence of exhaustion of the heart-muscle, and is a signal and a warning which the patient will neglect at his peril.

Exercise and effort are good up to the point at which discomfort is experienced, but on the first sensation of tightness across the chest, actual pain, or shortness of breath, he should instantly cease whatever effort he is making; he should never continue until the pain becomes disabling. His life must be reorganized on a lower level of effort, both physical and mental. He must lead a more leisurely existence. If there are causes undermining the stability of the nervous system, they must be removed. Business responsibilities must be lessened. Full dosage with ammonium bromide is helpful in removing restlessness and nervous irritability and in predisposing to sleep. Chloral hydrate and chloralamide are also serviceable in this direction.

In all patients liable to angina the diet should be carefully supervised so as to obviate any tendency to flatulence or dyspepsia.

When the blood-pressure is raised an occasional mercurial purge and a morning saline, sufficient to ensure at least one loose motion, are valuable.

The diet should be simple, light, and restricted in amount, and only a small quantity of fluids taken with each meal. It is better to omit rich soups and gravy. None but very slight exertion should be made immediately after a meal.

If syphilis is suspected or admitted and the Wassermann test is positive, antisyphilitic remedies may be administered.

Those patients who find that exposure to cold excites an attack should take every precaution to minimize this risk; they will probably find that chewing a tablet of trinitrin before leaving the house will materially help them. Cold baths and sitting in cold or draughty rooms should be avoided.

Potassium iodide, alone or combined with arsenic, and given over a long period, is of service in the arterio-sclerotic type of case.

The acute pain which sometimes accompanies an attack of paroxysmal tachycardia or the rapid action of auricular flutter and auricular fibrillation yields rapidly to morphia, which should not be withheld if the pain is great. As the paroxysm of rapid heart-action subsides under appropriate treatment and the cardiac rhythm becomes normal the pain will disappear.

In all cases of angina the essential treatment

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is to persuade the patient to live a rational and useful life well within his limitations, and to relieve symptoms as they arise. JOHN HAY.

ANGINA, VINCENT'S (*see* TONSILLITIS, ACUTE; DIPHTHERIA).

ANGIOMA (*see* NÆVUS).

ANGIO - NEUROTIC ŒDEMA (*see* ŒDEMA).

ANGULAR CURVATURE (*see* SPINAL CARIES).

ANISOMETROPIA (*see* REFRACTION AND ACCOMMODATION, ERRORS OF).

ANKYLOBLEPHARON (*see* EYELIDS, AFFECTIONS OF).

ANKYLOSTOMIASIS (*see* INTESTINAL WORMS).

ANOREXIA (*see* STOMACH, FUNCTIONAL DISORDERS OF).

ANTERIOR POLIOMYELITIS (*see* POLIOMYELITIS, ACUTE).

ANTE-PARTUM HÆMORRHAGE (*see* HÆMORRHAGE, ANTE-PARTUM).

ANTHRAOSIS (*see* PNEUMONOCOCCOSE).

ANTHRAX (*syn.* Malignant Pustule, Wool-sorters' Disease, Charbon (French), Milzbrand (German).—An acute specific infective disease, malignant in character, and producing either characteristic local lesions with a marked tendency to general systemic invasion, or a general systemic infection without visible primary local lesion, but accompanied by grave constitutional symptoms and usually fatal.

Etiology.—Anthrax is primarily a disease of the lower animals, more especially the herbivorous animals, the carnivora being far less susceptible. It is communicated to man by contact either with animals, or with animal or other material, containing the bacillus of anthrax or its spores. Thus it may be found among farmers, shepherds, cattlemen, veterinary surgeons, butchers, tanners, workers in wool and hair, horns, bones, etc., also among plasterers, dock labourers, furriers, brush makers, felt makers, and mechanics handling or repairing machines used in any of these industries. Of late years a number of people have been infected by means of spore-laden shaving brushes made from bristles imported from the Far East.

Admission to the human body is usually obtained by way of the skin, the continuity of which has become broken. It may, however, pass through the unbroken skin, especially when aided by friction accompanied by a greasy condition of the skin. It may conceivably also enter by the hair-follicles or sweat-ducts. In many factories the heat and moisture incidental to the processes of manufacture cause constant perspiration among the workmen; this softens the cuticle and probably increases the liability to infection. The dried spores contained in the dust of factories may be inhaled and give rise to an internal local infection, or may pass into the alveoli and be taken up by the wandering and epithelial cells and conveyed to the lymph-vessels in the alveolar framework, whence they pass to the glands at the roots of the lungs, later leading to general infection. Gastro-intestinal infection in man is rare, and when found is usually secondary to primary infection elsewhere, as when the serous discharge from cutaneous anthrax of the face runs into the mouth and thus carries bacilli into the alimentary tract.

Pathology.—The passage of *B. anthracis* (Plate 5, Fig. 5, facing p. 148) or its spores through the epidermis into the cutis vera sets up an acute though minute inflammatory focus in the subpapillary vascular plexus. Here the bacilli multiply and spread along the papillary bodies to the epidermis above, producing vesiculation in about twenty-four hours. A rapid effusion of sero-fibrinous lymph takes place into the cellular tissue and soon coagulates there, leading to necrosis of the central part of the invaded area. Thrombosis of the surrounding vessels simultaneously takes place and tends to inhibit general infection by sealing up the avenues by which access to the general circulation could be obtained. The specific toxins pass by osmosis into the surrounding tissues and lead to an excessive interstitial œdema of the surrounding skin, often at a distance of 12-15 in. from the focus of infection. In the surrounding tissues punctiform hæmorrhages may be seen. Although, as a rule, the anthrax bacilli do not penetrate to the subcutaneous tissues in the case of cutaneous infection, they frequently succeed in gaining entry to a blood-vessel not shut off by thrombosis, or they may penetrate the thrombus and reach the open vessel beyond, or, as more frequently happens, they may enter the lymph-vessel and be carried away in the lymph-stream. In either of these ways a general systemic infec-

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tion may be set up, with all the usual symptoms and pathological changes incidental to it. When local infection takes place in the mucous membrane of the alimentary or respiratory passages, a similar series of pathological changes and a similar sequence of events, *mutatis mutandis*, may be observed.

Post mortem, decomposition phenomena quickly show themselves, accompanied by rapid discoloration of the skin. This begins immediately after death, and in a few hours all the dependent parts of the body are of a dark purple colour. The lips, neck, and pectoral regions show this discoloration, and the ears, finger- and toe-nails, and penis and scrotum become almost blue-black. When there has been cutaneous anthrax the eschar is black and surrounded by a dark-coloured zone of skin. Bloody fluids usually exude from the mouth and nostrils, and are of a brownish or chocolate colour. On raising the breast-bone the cellular tissues are seen to be emphysematous in the substernal space, and bubbles of gas can readily be seen in the intercellular spaces. The pleural cavities contain large quantities of yellow clear fluid, the right side usually having more than the left. Occasionally this fluid is bloodstained. Collections of yellow gelatinous material are usually to be found under the pleura covering the lungs. The lungs themselves are engorged with dark blood, especially in their lower lobes. Distinct pneumonic consolidation is not usual. The glands at the roots of the lungs, which are greatly enlarged, frequently contain masses of intraglandular bloodclot. In the pericardial sac there are usually 4 or 5 oz. of serous fluid, and here again minute hæmorrhages on the pericardium and endocardium are common. The heart-muscle is dark-coloured and flabby. Less commonly, the abdominal cavity also contains a considerable quantity of fluid. Hæmorrhages may be noticed under the peritoneal coverings of the viscera, and here also subserous collections of gelatinous material are common. The spleen is usually large, dark-coloured, and diffuent, but it may be little changed. The kidneys and liver may show fine punctiform hæmorrhages. In cases where cerebral symptoms have been noted during life, extensive hæmorrhages are usually found in the subarachnoid space or under the pia mater. The whole surface of the brain may thus be obscured by a thin layer of dark-red blood completely levelling over the sulci. Hæmorrhages into the ventricles and also into

the brain substance itself are occasionally met with. If the post-mortem is made within twenty-four hours after death, cultures of *B. anthracis* can be made from all the cavities and viscera showing pathological changes.

Symptomatology.—In cutaneous anthrax the earliest symptoms are a slight burning or itching sensation, followed in a few hours by the formation of a small, flat, pale-red papule. Within twenty-four hours it is capped on its summit by a vesicle or group of coalescing vesicles. These are very shallow and flat, and are of a pale pearly or waxy appearance. The whole may be surrounded by a very faint red areola, and then closely resembles an ordinary vaccination mark about the third day. Usually, even at this early stage, a slight œdema of the surrounding skin is noticeable. The "malignant pustule," as yet, is not larger than a split pea and is painless. It is easy and most important to make a diagnosis, but, unfortunately, at this stage it is rarely recognized, and is thought by patient and doctor alike to be "nothing but a pimple." Even now there are, frequently, slight headache, a tendency to nausea, and a slight rise of temperature to 100-1° F. in the mouth, but often a couple of degrees higher in the rectum. The pulse may be a little faster than normal, perhaps 80-100 per minute, but otherwise normal. Within the next twenty-four hours the central vesicles dry up, become depressed, and assume a dark colour; while the vesicular area is gradually increased in size by the formation of fresh vesicles at its periphery. The constitutional symptoms grow worse, the temperature perhaps reaching 104°, and rigors may be present. If untreated, this area may gradually increase in size till it becomes even 2 in. in diameter, but it is not usual to see cutaneous anthrax covering an area larger than a shilling. Frequently, after attaining this size, it ceases to enlarge, dries up, and undergoes spontaneous cure, the slough separating in the ordinary way. As a rule, in industrial anthrax there is not more than one original focus of infection unless the inoculation has taken place by means of an infected nail scratch, or some similar way, when a row of "pustules" may result. The infection is usually from dried spores, and the symptoms are as described.

When infection is by the mature bacillus, as in butchers inoculated when slaughtering, the symptoms are somewhat different. Multiple lesions are more common, and the course is more rapid as well as more benign. There

ANTHRAX

is a greater tendency to the formation of large bullæ containing dark bloodstained fluid. The characteristic œdema is a very marked symptom when the lesion is near the orbit. The whole face may swell so enormously as to block up the various orifices almost entirely.

In **internal anthrax** the constitutional symptoms are similar to those in cutaneous anthrax, which, as already shown, are not very severe, and are due to absorption of toxins. In addition, however, are the symptoms peculiar to the internal organ in which the primary focus is situated. Thus in *gastro-intestinal anthrax* there may be abdominal pain, vomiting, or diarrhœa, the latter being occasionally hæmorrhagic. In *pulmonary anthrax* (wool-sorters' disease) there may be few localizing signs beyond perhaps a slight cough, and auscultatory and percussion examination may fail to reveal anything abnormal. When, however, in any of the forms infection becomes general, very severe constitutional symptoms at once manifest themselves. The pulse becomes faster and weaker, and this deterioration progresses with great rapidity. The respiration grows difficult and hurried, and may reach 40-50 respirations per minute. Cyanosis shows itself on the ears, lips, fingers, and toes. Râles become audible in the chest, and soon evidence of rapidly increasing pleural effusion is obvious. There is usually little or no expectoration, but when there is it may be bloodstained. If cerebral hæmorrhages are taking place, delirium and coma may supervene, or convulsions may occur. If the cerebral ventricles are the site of the hæmorrhage, rigidity of arms or legs may result. The temperature frequently reaches 104-6° F. shortly before death, which sometimes occurs with great suddenness from heart failure due to toxæmia. The condition is usually fatal within forty-eight hours of the onset of general infection. In some cases the toxæmia is so great that death takes place before the patient has experienced any alarming subjective symptoms.

Diagnosis.—The occupation of the patient may give valuable help in suggesting a diagnosis. In the case of **cutaneous anthrax** the diagnosis may be made almost at sight. The usual textbook description and illustrations of "malignant pustule" portray the disease at much too late a stage. One should aim at detecting the condition when it is only a few hours old, for by doing so the mortality is reduced at least 50 per cent. The important point to remember is that one should be on

guard whenever the patient's occupation suggests the possibility of anthrax, or when anything resembling the description already given is seen, and, when there is the least doubt, make a microscopical examination for the anthrax bacillus. The blood or serum for examination should be taken from the inner margin of the vesicular area. In the centre, when an eschar is present, the bacilli may be dead and degenerating and often stain badly. If a stain is not at hand, one can be made by dissolving a copying-ink pencil in water. If possible, the microscopical examination should be verified by a culture. In some *deep-seated boils*, at an early stage a papule forms with vesicles on its apex, and may then closely resemble cutaneous anthrax. Here the microscope would not show anthrax bacilli, and if a deep puncture were made into the centre of the papule it would probably yield a small quantity of pus—which is sufficient to exclude anthrax, as pus is never present in early anthrax. Anthrax is not painful, whereas a boil is painful. There are no pathogenic bacilli likely to be confounded with those of anthrax. Several cadaveric bacilli resemble it closely microscopically, and *B. subtilis* from hay is extremely like it also, but with care these should not cause any confusion.

In **internal infection**, as a rule, it is not possible to make a diagnosis in the early stages. In the later stages it can be done only by the exclusion of other more easily recognized conditions and by noticing that the great weakness of the heart's action and the general prostration are out of all proportion to any discoverable local lesion. Anthrax bacilli are not, as a rule, capable of detection in the blood earlier than twenty-four to thirty-six hours before death. In intestinal anthrax it may be possible to isolate the bacillus by means of a plate culture from the stools. Methods of blood and serum diagnosis by means of the opsonic index, agglutination, complement fixation, and precipitins may prove to be useful in the future, but, up to the present, have given but little help.

Prognosis.—In external anthrax the prognosis is distinctly hopeful, for at least 80 per cent. of cases recover under suitable treatment. The extent of the outward lesion, however, bears no relation to the gravity of the disease. In internal anthrax and general infection the outlook is as bad as it can possibly be, although a few cases of recovery after general infection have been placed on record.

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Treatment.—Early recognition, as we have said, is of the utmost importance. Having made the diagnosis, one has to distinguish between a case of external anthrax which is active and one which, having succumbed to the phagocytic action of the patient's blood, is undergoing spontaneous cure. This may be done by noting, in the latter case, the absence of fresh peripheral vesicles and of anthrax bacilli, or the presence only of degenerating bacilli which stain badly. If it is clear that spontaneous cure is well on the way, all that is required is the application of a moist compress soaked in 1 : 1,000 perchloride-of-mercury solution. If, on the other hand, active growth is evident, immediate local and general treatment will be urgently required, as at any moment general infection may ensue. The patient should be given an intravenous injection of 60 c.c. of *Sclavo's antianthrax serum*, and the local focus of disease should be excised. Excision is best done by means of Paquelin's or the galvano-cautery, cutting at least a quarter of an inch wide of the margin of the "pustule" all the way round and extending to the subcutaneous tissues below. It is then dressed *secundum artem* till healing takes place. This method obviates the risk of opening up fresh channels for infection, which is the disadvantage of cutting with an ordinary knife. If, however, a cautery is not available, excision may be made in the usual way with a knife, and the wound then thoroughly swabbed with 10-per-cent. carbolic acid, or even cauterized with pure carbolic acid, and afterwards left open to heal by granulation. The wound is occasionally washed with a weaker antiseptic and closed by immediate stitching, but there is some risk in doing so. Where excision is not possible or permitted, the area of the "pustule" may be injected at from five to ten points of its circumference, by means of a hypodermic syringe, with a 10-per-cent. solution of carbolic acid in equal parts of glycerin and water. The injection is made gradually as the needle is withdrawn in order to soak the tissues thoroughly with the solution. A compress of 1-in-1,000 perchloride of mercury is then applied. If, over the compress, an ice-bag be placed, it will also help to inhibit further growth of the bacilli. On no account should an ordinary poultice ever be applied to a "malignant pustule," as the heat and moisture proceeding from it encourage their further development.

If the case be one of general systemic infec-

AORTA, DYNAMIC PULSATION OF

tion or of internal local anthrax we must rely on Sclavo's serum alone, as it is the only agent from which we have much to hope at the present time. After the primary injection of 60 c.c. given intravenously, if urgent symptoms become manifest it may be repeated in twelve hours, and as often afterwards as the symptoms appear to demand. It may also be given intramuscularly four or five times a day in doses of 10-25 c.c. if preferred, after the initial dose of 60 c.c. has been given intravenously. The more rapid action of the intravenous method makes it preferable in the first instance. If the heart's action is becoming weak and the pulse rapid, benefit may be obtained by the use of digitalis. Patients should be kept under strict supervision for at least a week after the diagnosis is established. Nine or ten days after the use of the serum, "serum fever" frequently supervenes, accompanied by various skin eruptions and occasionally by arthritic symptoms.

WILLIAM MITCHELL.

ANTHROPHOBIA (see PSYCHASTHENIA).

ANTITOXINS (see IMMUNITY; PATHOLOGY, CHEMICAL).

ANTRUM, DISEASES OF (see SINUSES, ACCESSORY ATR, DISEASES OF).

ANURIA (see URINE, VARIATIONS IN AMOUNT OF; URINARY CALCULI).

ANUS, IMPERFORATE (see RECTUM, MALFORMATIONS OF).

AORTA, DYNAMIC PULSATION OF.—

In some cases the pulsation of the aorta may be very great, so much so that in the thorax it may be obvious, to the right of the sternum, both to the eye and the hand. At the same time there may be pulsation in the episternal notch, while the subclavians are higher than usual in the neck and throb violently. These phenomena are commonest in patients with regurgitation through the aortic valve, but may, on rare occasions, occur independently. Although, after death, the aorta appears of normal size, it is evident that it must have been dilated during life. The main importance of the condition is that it may be mistaken for aneurysm, but the pulsation is more diffuse, and aneurysm may at once be eliminated from the diagnosis by the use of the X-rays.

Similar excessive pulsation is not uncommon

AORTITIS, SYPHILITIC

in the abdominal aorta of young neurotic women who complain of dyspepsia. Aneurysm is very rare in this sex and at this age, and the pulsation has not its typical expansile character.

A. M. GOSSAGE.

AORTIC ANEURYSM (*see* ANEURYSM).

AORTIC INCOMPETENCE (*see* VALVULAR DISEASE, CHRONIC).

AORTIC REGURGITATION (*see* VALVULAR DISEASE, CHRONIC).

AORTIC STENOSIS (*see* VALVULAR DISEASE, CHRONIC).

AORTITIS, SYPHILITIC.—A localized mesoarteritis due to inflammatory changes in the vasa vasorum, accompanied by inflammatory changes in the adventitia and usually in the intima, and associated with the presence of the *Spironema pallidum*.

Pathology.—The infection is conveyed through the vasa vasorum. In acute cases the process begins in the media, but quickly involves the other coats; there are thickenings of the intima in the form of gelatinous plaques, at first soft and pink, later greyish and tough, associated with a corresponding area of periarteritis and mesarteritis. Shallow bluish depressions and puckerings may occur. Microscopically, there occur perivascular infiltration of the vasa vasorum, small-celled infiltration in areas of the intima and media, and degeneration and destruction of muscular or elastic fibres. Macroscopically, the process may be limited in extent, often localized at the root of the aorta. The process leads to acute aneurysm or acute dilatation, or to rupture.

Symptoms.—Pain is the most characteristic symptom. It may be merely a sense of constriction and oppression beneath the sternum, or burning in character, or of an alarming and agonizing kind in the region of the base of the heart, and may have all the characters of true angina pectoris. Its intensity is especially marked when the root of the aorta and the orifices of the coronary arteries are involved. It is accompanied by severe dyspnoea. Syncope and sudden death may follow. The attacks are not specially associated with exertion or cold, and they vary in duration. Dyspnoea occurs, and sometimes a condition resembling spasmodic asthma. The temperature is not usually raised. Physical signs, as a rule, obtain only when chronic disease of the aorta coexists. On the other hand, signs of aortic

APHASIA

dilatation and insufficiency may suddenly appear in one who was previously without signs of arterial disease. In chronic cases the symptoms are those of atheroma.

Diagnosis.—The coexistence of anginal and dyspnoic attacks together with the development of the signs of aortic dilatation, especially in a young adult previously without chronic arterial disease, would indicate acute aortitis.

Prognosis is very grave when aortic incompetence exists, also in acute aortitis, particularly when anginal attacks occur.

Treatment.—Mercury inunctions should be administered early, along with potassium iodide. In acute cases rest must be absolute; pain and distress may be treated by an ice-bag suspended from a cradle, or by morphia injections.

OLIVER K. WILLIAMSON.

APHASIA (including Alexia, Agraphia, etc.).

—Like all other cerebral symptoms, aphasia may be regarded from a double viewpoint—(1) anatomical and (2) physiological. Anatomically, we are concerned with the sites and the sorts of lesions that give rise to aphasia; physiologically, we examine the nature of the disturbance of cerebral function produced by them. The particular symptom, aphasia, moreover, presents (3) a psychological aspect for consideration which renders its complete study a matter of much complexity and not a little perplexity. Aphasia is a cerebral symptom produced by certain pathological lesions in certain anatomical areas, and exhibiting clinically certain types of physiological disturbance, the psychical side of which is not infrequently that which most attracts the examiner's attention. For the sake of lucidity the practitioner should endeavour to realize this many-sidedness of aphasia, and keep the terminology and ideas connected with each of these aspects from getting confused in his mind with those of any of the others.

To begin with the clinical side. There are certain types of aphasia well recognized and readily distinguishable when they occur in a pure form. Of these, one is known as motor aphasia (aphemia), the other as sensory aphasia. Each of these, further, occurs in a less complete form, known as pure (or subcortical) motor and pure (or subcortical) sensory aphasia respectively; also in a somewhat more complex form, known as transcortical motor aphasia and transcortical sensory aphasia respectively. The two main types, again, may be compounded into a further type—complete or total aphasia.

APHASIA

Then, finally, there are isolated disturbances of writing-speech, known as pure alexia and pure agraphia.

1. *Complete motor aphasia*.—Clinically, the patient cannot speak, or at most has only a few words or parts of words. He cannot repeat words, and he cannot read aloud. As a rule, also, he cannot write spontaneously or to dictation, but he can copy.

2. *Complete sensory aphasia*.—The patient does not understand what is said to him. He has the same difficulty in understanding what he reads as what he hears. He often has a certain difficulty in speaking, known as paraphasia or jargon aphasia; also in writing, known as paraphraphia.

3. *Complete or total aphasia*.—In the combined form the motor aphasia naturally masks the existence of the sensory aphasia, which, however, at least to begin with, can usually be demonstrated when it exists.

4. *Incomplete, pure, or subcortical motor aphasia*.—The clinical condition is one of pure "word-dumbness:" reading and writing are intact.

5. *Incomplete, pure, or subcortical sensory aphasia*.—Clinically there is pure word-deafness; hence, naturally, the patient cannot repeat words to order and cannot write to dictation. But he can speak, read, write, and copy.

6. *Transcortical motor aphasia*.—The patient cannot speak or write spontaneously, whereas he can repeat what he hears and can copy and write to dictation. A variety of this is the so-called amnesic aphasia or verbal amnesia.

7. *Transcortical sensory aphasia*.—The patient repeats in a parrot-like fashion what he hears, not knowing what it means. He can read aloud, can write to dictation and copy, without understanding what he reads or writes. His spontaneous speech is usually much reduced.

8. *Pure alexia*.—The patient cannot read.

9. *Pure agraphia*.—The patient cannot write.

The above varieties are well-recognized and differentiated clinical types, some of which are rare, while others are common.

Considering next the **anatomical** side of the speech mechanisms, we know that in right-handed individuals the speech centres are mainly located in the left cerebral hemisphere. Here there is unquestionably a speech area, lesions in any part of which are followed by one or other clinical variety of aphasia in an overwhelming number of instances. The exceptions only prove the rule. The area em-

braces the inferior posterior part of the frontal lobe, the island of Reil, that part of the precentral gyrus which overhangs the island of Reil (operculum Rolandi), the posterior part of the temporal lobe, and part of the inferior section of the parietal lobe. As for the question of subdivisions in this speech area, it is agreed by most observers that lesions in the frontal part, including the anterior portion of the island of Reil, cause motor aphasia (1), whereas lesions in the temporal and parietal section give rise to aphasia of a sensory type (2). A large lesion in the distribution of the Sylvian artery and its branches will give rise to complete aphasia (3). Subcortical lesions in the temporal part of the speech area may produce pure word-deafness (5). A lesion interrupting fibres passing between the posterior inferior extremity of the third left frontal gyrus and the Rolandic centres for the movements of tongue, lips, palate, etc., may give rise to pure motor aphasia (4). The transcortical varieties of aphasia, for reasons which need not be entered on in this place, are not likely to present that definite anatomical localization which we find in the case of the simpler varieties. It may be remarked, however, that transcortical sensory aphasia (7) has been seen in severe atrophic lesions of the temporal lobe. Deep lesions under the parietal part of the speech area may cause pure alexia, with or without an accompanying hemianopia (8).

Pathologically, the common causes of aphasia are cerebral embolism, hæmorrhage, thrombosis, inflammation (i.e. toxi-infective conditions), and tumours. These processes produce aphasia just as they may produce motor paralysis, and the reader is referred to the appropriate sections for further discussion of their pathology (see **CEREBRAL VASCULAR DISEASE, CEREBRAL TUMOUR**, etc.).

Psychologically, aphasia is but a part of a larger disturbance of function, which is concerned with recognition and expression. A person may recognize the different sense-qualities of an object, yet be unable to combine these into one conceptual whole; e.g. he may recognize, through different sense-impressions, the bark, trunk, boughs, twigs, leaves of a tree independently, yet fail thereby to reach the concept "tree" or to have the normal associations of the same aroused in his mind. Again, he may fail to recognize an object, e.g. a cake of soap, by one sense-avenue, such as sight, yet recognize it by its smell. These conditions are varieties of what

is called *agnosia*; the first is *ideational* agnosia, the second *sensory* agnosia. Now, when a patient sees written words and recognizes that they are words, but does not know their meaning, he is suffering from agnosia for written language. Sensory aphasia is only a special part, on its sensory side, of agnosia.

Similarly, *apraxia* (q.v.) is the term employed to signify inability to perform certain movements in the absence of actual paralysis. Motor aphasia is only a special form of apraxia; it is apraxia of the musculature of the lips, palate, tongue, etc. The patient can employ the same muscles for eating, swallowing, etc., without difficulty.

Viewed in this light, the symptoms of motor and sensory aphasia fall into line with other psychomotor and psychosensory symptoms, and are seen not to possess any unique or unusual character differentiating them from all other disturbances of psychical function.

The symptom of dysarthria or anarthria is discussed in SPEECH, DISTURBANCES OF.

In any case of aphasia, motor or sensory, the **prognosis** is the first question that is sure to be propounded to the practitioner by the patient's friends. Unfortunately, owing to the necessity for taking into consideration quite a number of factors in any given case, the prognosis is often a matter of much complexity. The following generalizations—to which, needless to say, there are always exceptions—will be found to be of some practical value in this connexion.

1. Speaking generally, the older the patient, the less likely is he to make a good recovery. Yet one frequently meets with notable exceptions to this rule. Some children who develop aphasia after encephalitis never make that improvement which a consideration of their age would lead one to expect. Some cases in elderly or old people, on the other hand, do remarkably well.

2. The longer the interval between the stroke and the reappearance of speech, the less likely is speech to be abundant. Should the patient be able to articulate some words again within a few weeks, and still more if he can do so within a few days, the outlook is certainly more favourable than if a very long interval elapses. A case is on record, however, in which a patient of 54, after no less than six years of motor aphasia, began gradually to improve, acquired new words, and eventually could converse on ordinary subjects.

3. Much depends on the severity of the attack. If the aphasia is unaccompanied by paralytic symptoms the chances of functional restoration are better than if there is marked hemiplegia. To this rule also, however, there are discouraging exceptions.

4. If there is little or no indication of generalized cardiac, arterial, or renal disease the outlook is better than when there are degenerative changes in these systems.

It should always be borne in mind, in considering prognosis, that there is a large class of cases in which the aphasia is essentially transient; here are included cases of Jacksonian epilepsy, migraine, cerebral arteriospasm, incomplete thrombosis, and various toxi-infective conditions. It should be remembered, further, that in not a few cases the aphasic symptoms are derived from pressure (e.g. of a clot) and are not the result of actual destruction of tissue. Hence, with the removal of pressure some degree of functional restitution is to be expected. And, once more, the lesion itself may throw out of action indirectly, by shock or otherwise, certain functional mechanisms, and these may in due course recover completely, never having been structurally damaged. In a consideration of these points will be found the explanation of the varying prognosis in cases of aphasia.

Treatment.—In any given case we must first determine as far as is possible the nature of the underlying pathological condition, and proceed to treat that without delay. The most frequent diseases provocative of aphasia, as already mentioned, are embolism, thrombosis, hæmorrhage, tumours, and inflammatory or infective conditions. In one sense, aphasia is to be regarded as a symptom of such conditions, strictly comparable to the motor paralysis or any of the other paralytic phenomena that may ensue; and in that sense its treatment is that of the disease.

When, however, the aphasia is, as it were, established, and apparently stationary, the question arises whether compensation may not take place, and whether other parts of the cortex may not be educated to function for those that appear to be permanently affected. Without going into the details in this place, it may be said that adequate evidence has been brought forward to show that treatment, by methods of re-education is well worth pursuing, and that the comparative neglect of such methods by the medical profession is matter for regret.

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These methods may be briefly outlined as follows :—

1. *Repetition methods*.—The patient is taught afresh, as at school, to repeat simple nouns, verbs, and other parts of speech; he learns his letters again, and is taught to combine his words into sentences. When a noun is named, he at the same time is shown the object, and allowed to feel it, and he sees it written on a piece of paper before him. Thus visual, auditory, and tactile memories are stimulated at the same time. Reading aloud, copying, and writing from dictation are also included as the patient progresses.

2. *Phonetic methods*.—The patient learns the letter-sounds again, and is taught such sounds as papa, apap, appa, then baba, abab, abba, mama, amam, amma, and so on through the alphabet.

3. *Lip-reading method*.—He learns the movements of articulation, enunciation, and vocalization, either from observing the lips and tongue of others, or by observing himself in a mirror.

The best results in practice are obtained by a combination of these methods. Needless to say, as I have remarked elsewhere, "the combination of a suitable case, a diligent pupil, and an enthusiastic and patient teacher is almost a *sine qua non*, and doubtless such a combination is not always to be found." Yet even a modicum of success is worth striving for.

S. A. KINNIER WILSON.

APHEMIA (see APHASIA, p. 95).

APHONIA (see SPEECH, DISTURBANCES OF).

APHTHOUS STOMATITIS (see STOMATITIS AND GLOSSITIS).

APLASTIC ANÆMIA (see ANÆMIA).

APOPLEXY (see CEREBRAL VASCULAR DISEASE).

APPENDICITIS.—From the clinical standpoint the term appendicitis includes all acute and chronic inflammatory conditions of the vermiform appendix, together with those localized extensions of inflammation that may arise from the appendix as a centre.

Pathology.—At first sight the pathology of acute appendicitis appears extremely complicated, owing to the numerous varieties of inflammation that have been described, but when it is remembered that all the so-called varieties are merely different stages of the same process,

and are similar to what is met with in inflammatory conditions elsewhere in connexion with mucous tracts, all difficulty disappears.

The primary stage in all cases of appendicitis is probably catarrhal in nature (*acute catarrhal appendicitis*), and in some cases the process may not extend further. The mucous lining is swollen and oedematous, and pours out an excessive amount of its normal secretion. The vascular changes which attend inflammation are present, and even the subperitoneal vessels may be engorged. In the next stage, which is known as *acute diffuse appendicitis*, the inflammation has extended to all the coats of the appendix. The organ is swollen and rigid, and presents a dark-red appearance. Ulcers may be found in the mucous membrane, more especially in the neighbourhood of faecal concretions, and these may extend through the wall and be marked externally by yellowish necrotic areas, one or more of which may perforate (*acute perforating appendicitis*).

Purulent appendicitis is a further stage of the acute diffuse process. In mild infections suppuration does not occur, but if the infection is a severe one pus may form either within the cavity itself, when it may give rise to an endo-appendicular abscess, or within the appendicular wall. In the latter case small abscesses may be found projecting on the surface of the appendix and by extension a localized peritoneal abscess or acute diffuse peritonitis may be set up.

In *gangrenous appendicitis* the whole or a part of the appendix sloughs. In appearance the gangrenous area is black and swollen, and to the touch soft and friable. Gangrene may occur, primarily due to a twist of the meso-appendix or a bend which obstructs the blood supply, but more frequently it is the result of a severe inflammation which rapidly produces a spreading thrombosis of vessels. It is usually associated with some obstruction of the lumen of the appendix.

Chronic appendicitis results from repeated acute attacks. The appendix itself is thickened and rigid, and on making a cross section the lumen does not collapse, but remains patent. The mucous membrane may be partly replaced by fibrous tissue, and there is much formation of fibrous tissue throughout the muscular coats. Adhesions are frequently present in the neighbourhood of a chronically inflamed appendix, and account in part for the symptoms.

Obliterative appendicitis.—This name is ap-

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plied to the terminal stage of many cases of both acute and chronic appendicitis. When the mucosa is acutely inflamed, desquamation of the epithelium takes place and the denuded areas, by adhering to one another, may lead to complete obliteration of the lumen. If this occurs throughout the entire length a natural cure of appendicitis may thereby result. In many cases, however, obliteration occurs over limited areas only, and when this obliteration is situated above the actual apex of the organ, the distal portion, which is already the seat of inflammatory changes, may become dilated into a cyst containing mucus or pus. This variety of cystic appendicitis may readily develop into the acute perforating variety, and is also a common condition met with in so-called *relapsing appendicitis*.

Peritonitis may occur in any attack of appendicitis involving the peritoneal covering, and may be either (a) localized or (b) diffuse.

(a) In some very acute cases a *localized* serous or serosanguineous exudate is found, limited to the periappendicular region, though not surrounded by any adhesions. The more common local extension, however, consists in the formation of an *abscess*. Its position will vary with that of the appendix, but, speaking generally, it is usually in the neighbourhood of the right iliac fossa. Some pelvic abscesses lie so deeply as to present no signs on examination of the external abdominal wall, and the same remark applies to small abscesses situated among the intestinal coils internal to the cæcum, and to some retrocæcal abscesses. Most cases, however, present unmistakable clinical signs in the appendix region. Periappendicular abscesses contain, as a rule, foul-smelling pus swarming with the *B. coli communis*.

(b) *Diffuse* peritonitis, secondary to appendicitis, differs little in its pathology from other varieties of spreading peritonitis and, in consequence, need not be further considered.

Etiology.—Appendicitis is far more common in early than in late life, statistics showing that by far the largest number of cases are met with between the ages of 10 and 30. It must not be forgotten, however, that it may occur in quite young children, and is perhaps more common in them than is generally supposed, and that it may also be met with even in extreme old age. The greater liability in early life to the disease is probably accounted for by the greater activity of the lymphoid tissue at that age. It is commonly believed, now, that the appendix is not purely a vestigial

organ, but that its highly developed lymphoid apparatus serves as a protection against bacterial attacks, from which it is itself prone to suffer. In later life the lymphoid tissue here, as elsewhere, undergoes atrophy, and the appendix is therefore less liable to suffer. More males are attacked than females, possibly because of their greater liability to exposure and to dietetic errors.

Although the actual immediate cause is bacterial, it is necessary to refer to some *predisposing causes*. For many years foreign bodies were regarded as a potent cause, and this belief is justified by more recent observation, which has, however, shown that the foreign bodies are usually formed *in situ*. The normal appendix, save for the presence of a small amount of mucus, contains nothing in its lumen. In diseased conditions concretions are frequently found. These concretions are primarily formed by a precipitation of the calcium salts normally present in the appendicular mucus. Once formed, the concretion tends to grow by the laying down of layers of inspissated mucus and by the accretion of small faecal particles. Quite plainly, a concretion predisposes to inflammation, partly by its direct pressure on the mucous membrane, partly by interference with blood supply, and partly by obstructing the emptying of the appendix, thereby leading to cystic dilatation. Rarely, actual foreign bodies may be found—grape-stones, gall-stones, pins, grains of shot, intestinal parasites, more particularly *Oxyuris vermicularis*, and the like—but the rôle played by these is comparatively unimportant.

Several anatomical factors also serve as predisposing causes of inflammation, such as the narrowness of the lumen, the single supplying artery, and the abundant lymphoid tissue. The frequency with which appendicitis and tonsillitis occur in the same individual has often been noted, and is probably due to the fact that the lymphoid tissue throughout the body is liable to succumb to the attacks of certain micro-organisms. In the case of the appendix the infection may, as Widal suggests, be blood-borne.

The appendix also suffers occasionally in certain specific fevers. The evidence is not precise, but is strong in favour of influenza being a causal factor. Rheumatic fever also is regarded by some as an important cause, while enteric fever, without doubt, is accompanied by changes in the lymphatic tissues of the appendix, as is also bacillary dysentery.

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Occasionally, direct trauma has preceded an acute appendicitis.

Bacteriology.—By far the most frequent organism found in connexion with both suppurative and non-suppurative appendicitis is the *B. coli communis*. Whether it is often the primary cause is a matter of controversy, some authors believing that its frequent presence is mainly due to its vitality and power of overgrowing other pathogenic organisms. Whether this is so or not matters little, as the organism is undoubtedly extremely virulent in most of the cases in which it is present.

According to Dudgeon and Sargent, streptococci are seldom present. Staphylococci are more often found, both the *Staphylococcus albus*, whose pathogenicity is slight, and the *Staphylococcus aureus*. Other organisms have also been found, such as the pneumococcus, the *B. pyocyaneus* (not necessarily associated with blue or green pus), and *B. prodigiosus*. Some writers lay great stress on the importance of anaerobic organisms, but recent investigations have failed to prove either their frequent presence or their importance.

Symptomatology.—This will be discussed under the following heads:

1. Acute uncomplicated appendicitis.
2. Acute appendicitis with localized abscess formation.
3. Acute appendicitis with diffuse peritonitis.
4. Chronic appendicitis, including appendix dyspepsia.
5. Appendicitis in children.

1. **Acute appendicitis.**—It cannot be too strongly emphasized that very severe appendicitis may exist with comparatively slight symptoms and local signs. As a rule the diagnosis is easy, but sometimes may be almost impossible. The following symptoms and signs are usually present. (a) **Pain.**—Most attacks start suddenly with abdominal pain. At first this may be felt all over the abdomen, or more especially at or around the umbilicus. After a time the pain may become acute in the neighbourhood of the right iliac fossa, but this is by no means constant. Sometimes it is more gradual in onset. It is in all cases severe and, when situated in the right iliac fossa, may cause the patient to assume the dorsal position with the right thigh partly flexed on the abdomen. (b) **Vomiting** often occurs with the first onset of symptoms, but is seldom prolonged unless diffuse peritonitis rapidly ensues from a perforated or gangren-

ous appendix. At the beginning there may be a little diarrhoea, and, indeed, this symptom may precede the actual onset of pain, though previous constipation is more common. Later on, constipation is the rule, and in severe cases may be accompanied by tympanitic distension of the abdomen. (c) Some **irritability of the bladder**, giving rise to frequency of micturition or pain during micturition, is common. It may be due to the appendix lying in the pelvic position in close proximity to the bladder, but is met with even when the appendix is retrocaecal. On the other hand, it may be entirely absent. (d) **Rise of temperature** of 1–3 degrees above normal is to be expected, but no great reliance can be placed on this sign. It is undoubtedly met with in the majority of cases, but is very variable, and in some of the most severe cases the temperature may be subnormal. Even with abscess formation or commencing diffuse peritonitis the temperature may remain normal. In fact, for diagnosis and for an estimation of the actual severity of an undoubted case, more reliance must be placed on the phenomena as a whole, including the general aspect of the patient, than on any single sign. (e) **Pulse.**—More stress can be laid on the condition of the pulse, yet it is not always a reliable indication. Increased rapidity is almost the rule, and great acceleration generally points to a severe infection. A fast, irregular pulse is a sign of great gravity. (f) **Tenderness** on palpation is one of the most valuable diagnostic signs. Sherren has called attention to the importance of cutaneous tenderness elicited by gently pinching or pricking the skin over the area supplied by the 11th dorsal nerve, and immediately above and below it. More important, however, is the tenderness made out by firm pressure over the area around McBurney's point, i.e. a point $1\frac{1}{2}$ –2 in. from the right anterior superior spine of the ilium along a line drawn from that spine to the umbilicus. Sometimes the pain is felt most acutely when the pressure of the examining hand is suddenly withdrawn (Blumberg's sign). When pain is absent or slight over McBurney's point,¹ pressure should be made at the back, about the middle of the iliac crest, as in some cases of retrocaecal appendicitis a distinctly painful area may be

¹ Some writers believe that Clado's point, situated at the point of intersection of the right rectus and a line joining the two anterior superior iliac spines, is a more accurate guide to the base of the appendix than McBurney's point.

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detected where the lateral cutaneous branches of the lower dorsal nerves approach the surface. In no case should a rectal examination be omitted, as not only may the swollen appendix, or a localized bulging due to an abscess, be felt, but even in the earliest cases a tender point may be found high up on the right side of the rectum. (g) *Rigidity*.—Some resistance to pressure caused by spasm of the lower part of the right rectus muscle is the rule. This sign is seldom absent when pain and tenderness are present, and it is most valuable in diagnosis. When pronounced, it is an almost certain sign of extension of the inflammation to the peritoneum. In cases of pelvic appendicitis it may be very slight. (h) *Tumour*.—It is seldom possible to palpate the appendix itself, but this may occasionally be done when it lies anteriorly and there is not much muscular rigidity. As a rule, however, an ill-defined swelling can be detected in the right iliac fossa. This is partly caused by spasm of the cæcum, and partly by œdema of the cæcal wall and the periappendicular tissues. Some dullness on percussion may be made out over this swelling, but, in the early stages, this is by no means constant. Ewart lays stress on the importance of dorsal percussion over the sacro-iliac joint, and states that dullness in this area invariably points to appendicular trouble. This sign is certainly obtainable in many cases, but is often absent.

In addition to the symptoms and signs already enumerated, rarer ones may be encountered. Hæmatemesis is occasionally met with; it may be severe, and is probably due either to the action of toxins on the walls of the gastric vessels or to their involvement in a spreading thrombosis of the omental veins. Chills sometimes occur, but are most often present when an abscess has formed. Jaundice may be seen, due either to a catarrhal cholangitis or to a complicating suppurative pylephlebitis, and lastly distension of the superficial circumflex iliac vein may be noted as an early sign.

To sum up, pain in the region of the right iliac fossa, accompanied by local rigidity and tenderness, with mild fever and general digestive disturbance, constitute the main phenomena of acute uncomplicated appendicitis. In deciding as to the severity of any given case the general aspect of the patient must be considered and the symptomatology as a whole, as any individual sign may be misleading or absent.

2. Acute appendicitis with localized abscess-formation.—In the first stage of acute appen-

dicitis it is quite impossible to determine whether the process will go on to abscess-formation or not. In cases which terminate by resolution the symptoms usually begin to subside in twenty-four to seventy-two hours, but sometimes cases will drift on in a subacute condition for a week or more and then completely clear up.

If an abscess forms, the general symptoms, after a few days, will usually become worse; the temperature reaches a higher level, the pulse remains fast, rigors and sweating may occur, and the local signs become more pronounced. On examination a distinct swelling is now palpable, and is more definitely circumscribed than before; pressure over it causes great pain. Fluctuation may be detected, and in neglected cases œdema, and even redness of the abdominal wall, may be present. The actual signs will, of course, vary with the situation of the abscess. If it is situated among the coils of small intestine the physical signs will often be few, and if in the pelvis it may be detected most readily by rectal examination. In doubtful cases a blood-examination may give valuable assistance. In the early stage of most attacks there is a leucocytosis of from 15,000 to 25,000 per c.mm., the actual height of the count varying inversely with the severity of the inflammation. The only exception to this statement lies in the fact that in the most severe variety of gangrenous appendicitis the white-cell count may be low. Here, however, the other clinical features should lead to a correct diagnosis. If the count remains high or is still increasing after the lapse of three to four days from the commencement of an attack, it may be regarded as evidence in favour of an abscess.

Abscesses untreated by an operation may rupture in various situations. The gravest accident that can occur is rupture into the general peritoneal cavity, an event usually attended by sudden intense abdominal pain and collapse, and followed by all the symptoms of a rapidly spreading and almost invariably fatal peritonitis. Rupture into the cæcum, ascending colon, or a coil of small intestine may occur, and is often followed by an uncomplicated recovery. Other situations where rupture may take place are the rectum, the bladder, the vagina, and very rarely on to the abdominal wall.

Occasionally abscesses may remain almost entirely latent, and it is not uncommon, when operating after an attack of appendicitis when

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has apparently completely subsided, to find the remains of inspissated pus.

3. Acute appendicitis with diffuse peritonitis.

—Diffuse peritonitis may result almost immediately by direct spread from an acutely inflamed appendix, or, as indicated in the previous section, may follow rupture of a localized abscess. It is its liability to occur as a primary condition that makes the careful watching of all cases of appendicitis from the onset so important, and that renders immediate operation an imperative duty in all cases of doubt. When once fully developed, peritonitis of appendicular origin does not differ in its features from other varieties of diffuse peritonitis. Symptoms of general toxæmia are present; the expression is anxious, the colour muddy, the eyes are sunken, the tongue is dry and furred, and vomiting is often persistent. The pulse is usually fast, and the temperature raised, but neither pulse nor temperature is an infallible sign. In the very worst cases the temperature may be subnormal and the pulse under 100 till near the end. The patient lies in the dorsal position with legs drawn up, and with costal respiration. The abdomen is swollen, the actual degree of tympanitic distension depending on the duration and severity of the inflammation. The abdominal wall is rigid and board-like, and tender all over to touch and to percussion. Sometimes evidence of free fluid may be obtained by finding movable dullness at the flanks. Local signs in the right iliac fossa may be detected, but are often lost in the general peritoneal signs. Diarrhoea may occur, or, on the other hand, obstinate constipation, and in the last stages hiccough is a troublesome and ominous sign. When this picture is seen, diagnosis is obvious, but it cannot be too strongly insisted that, at the beginning, the symptoms may be totally misleading.

4. Chronic appendicitis. — The symptoms caused by chronic appendicitis are most variable. In some subjects no evidence whatsoever implicating the appendix may be found, and yet on opening the abdomen adhesions and chronic inflammation of the appendix may be discovered, and removal of the organ is followed by the disappearance of various symptoms of alimentary disturbance. It is to these cases that Moynihan has given the name "appendix dyspepsia," a form of dyspepsia which he describes as capricious. Pain in the stomach is experienced after food, but the interval is variable; the pain is worse after exertion, and may even be accompanied by hæmatemesis and

epigastric tenderness. Flatulence and some eructations may be noted, and as a rule there is hyperchlorhydria, but in a percentage of cases hypochlorhydria is encountered. Undoubtedly cases of this sort occur, even if they are less common than is now supposed. The dyspepsia is to be explained partly as a toxic and partly as a reflex nervous disturbance. In other cases frequent attacks of mild pain in the right iliac fossa are complained of, and there may even be persistent dragging pain shooting down into the thigh and scrotum. These patients may suffer frequently from mild febrile attacks, and may have symptoms of mucous colitis. Violent exercise, exposure to cold, and dietetic errors may increase the symptoms.

Associated with symptoms of chronic appendicitis is a condition that is now commonly spoken of as *Jackson's membrane*. In these cases there is a veil-like layer of peritoneum stretched across the cæcum and ascending colon for a variable distance, sometimes extending up to and even involving the hepatic flexure; it may or may not be associated with adhesions and other signs of inflammation around the appendix itself. Sometimes it is evenly spread over the intestine as a thin, cobweb-like veil, blending on the inner side with the great omentum or with the peritoneum covering the posterior abdominal wall, and on the outer side with the peritoneum of the lateral abdominal wall; at others it appears collected in the form of one or more bands stretching across and more or less definitely constricting the cæcum, ascending colon, or hepatic flexure. It is often associated with dilatation and abnormal mobility of the caput cæci. Opinion is divided as to its origin, one explanation being that it is due to inflammation and is therefore comparable to adhesions formed elsewhere in the abdomen. According to other observers, it is congenital in origin, and is really the thinned-out lateral margin of the omentum, which spreads over the hepatic flexure, and fuses with the posterior parietal peritoneum to the outer side of the ascending colon; during the descent of the cæcum it becomes adherent to the peritoneum covering the anterior surface of the colon and cæcum. In all cases of chronic appendicitis the presence of this membrane should be looked for, and when found it should be removed if possible, either by dividing it along the anterior surface of the cæcum and ascending colon, or by dividing bands if they are present. When

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well developed the bands may have to be ligatured, and in all cases raw surfaces should be covered, as far as possible, either by purse-string sutures or by omental grafts.

Diagnosis of chronic appendicitis is often difficult and, when localizing signs are absent, may be impossible. Even when apparently local signs are present an operation may disclose a perfectly normal appendix, and the symptoms may prove to be the result of visceroptosis, or due to obstruction to peristalsis in the ascending colon caused by a Jackson's membrane. In other cases kinking of the terminal part of the ileum may account for the uneasiness in the right iliac fossa. Bastedo has described a sign which may possibly give aid in doubtful cases. He states that when the colon is distended with air by means of a long tube passed into the rectum, distinct pain will be produced in the appendix region if chronic appendicitis or appendicular adhesions exist. Aid may also be obtained in diagnosis by means of radiographic examination following the administration of a bismuth meal or the injection of bismuth into the colon.

5. Appendicitis in children.—It has only of late years been recognized that appendicitis is quite commonly met with in children. It is rare in infancy, infrequent under 5 years of age, and comparatively common between 5 and 15. Many writers have pointed out that so-called "bilious attacks" in children, consisting of abdominal pain, headache, vomiting, languor, and malaise, are frequently appendicular in origin. Every case of "bilious attack" should, at any rate, be held suspect, and kept under the closest observation. Localized abscess-formation and spreading peritonitis are both extremely common, but if the practitioner is on his guard it should be possible to diagnose the condition in most cases before the occurrence of such an eventuality. The presence of abdominal pain and vomiting is itself significant, if accompanied by a rise of temperature and pulse-rate. There is usually fixation of the abdominal wall, and diarrhoea is more frequently met with than in adults. Frequent micturition is also a common symptom. In most cases examination, if carefully carried out, will reveal localizing signs of the same nature as have been described in the adult, but the absence of these should not lead to a delay in operating if the general symptoms are severe and persistent.

Complications of appendicitis.—The most important complications—namely, local-

ized abscess-formation and diffuse peritonitis—have already been dealt with. Either condition may be associated with severe ileus and abdominal distension. Other complications are—(1) Jaundice, due to catarrhal cholangitis, or to portal pyæmia; in the latter condition pyæmic abscesses form within the liver, and the general condition of the patient rapidly becomes very grave. (2) Thrombosis of the portal vein, and more rarely mesenteric thrombosis. (3) Thrombosis of the external iliac vein; this complication is not uncommon, and is usually recovered from. (4) Subphrenic abscess, which may arise by direct extension, or because of an abnormal situation of the appendix; it may also result in the course of general pyæmia. (5) Suppurative pleuritis, and abscess in the lung as a part of a general pyæmia. (6) Salpingitis and other pelvic inflammations may occur in women.

Differential diagnosis.—It is almost impossible to enumerate all the conditions that have been mistaken for appendicitis. In the majority of cases a careful examination and due attention to the history and symptoms of the illness, will prevent mistakes, but sometimes immediate diagnosis is impossible. The following conditions must be borne in mind in arriving at a conclusion :—

1. Perforation of the stomach or intestine.—In many cases the extravasated contents of the stomach escaping from a ruptured pyloric ulcer pass downwards into the right iliac fossa, and give rise to definite pain and other symptoms in the lower right quadrant of the abdomen. The knowledge that this may occur, the history of the case, the existence of early collapse and general tympanites, should help to a correct diagnosis. In ambulatory typhoid a perforation at the lower end of the ileum may also simulate an acute appendicitis.

2. Intestinal obstruction.—As a rule there will be no localizing symptoms in the right iliac fossa, and the absence of rise of temperature, in spite of the existence of severe abdominal symptoms pointing to obstruction, should exclude appendicitis. In cases of intussusception the sausage-like tumour and the passage of bloody mucus by the bowel are practically pathognomonic. In some cases, however, the practitioner must be content to diagnose an "acute abdomen" requiring immediate operation. If this is correctly done, the patient stands a much better chance of recovery than if time is wasted in trying to arrive at an exact diagnosis.

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3. *Female pelvic diseases.*—In women septic salpingitis, a ruptured tubal pregnancy, a twisted ovarian cyst, and the like, must be considered. A pelvic examination, if necessary under an anæsthetic, will generally enable a correct opinion to be arrived at.

4. *Intestinal colic.*—The pain of intestinal colic, or of acute indigestion, may sometimes be extremely severe, and it is at times impossible, at first, to exclude the existence of an acute inflammatory lesion. The absence of rise of temperature, the existence of an adequate cause for the colic, and the rapid subsidence of symptoms are important differentiating signs. In cases in which the least doubt exists, opium should on no account be given. It has been well stated that a good physician is known by the fact that he will not administer morphia in undiagnosed cases of abdominal pain.

5. *Biliary colic.*—In this condition the pain is usually situated in the upper portion of the abdomen, and rigidity, if present, involves the upper part of the right rectus muscle. A slight tinge of jaundice, the presence of bile in the urine, and the history of previous attacks, help the diagnosis.

6. *Renal colic.*—The situation and character of the pain, and the presence of blood in the urine, are the most important differentiating signs. In cases of *movable kidney* a sudden obstruction of the ureter may occur accompanied by intense pain, but, as a rule, in these cases a swollen and tender kidney will be felt lying at an abnormally low level in the abdomen.

More difficulty exists in diagnosing between movable kidney and chronic appendicitis, as both may be associated with obscure alimentary disturbance and with pain and tenderness in the lower abdomen. A consideration of the type of patient, and of the relief afforded by a supporting kidney truss, may lead to a conclusion, but it must be remembered that both conditions may easily coexist.

7. *Pleurisy and pneumonia.*—It is a matter of common knowledge that basal pleurisy or pneumonia may simulate acute abdominal disease, pain being referred to the abdomen along the course of the lower costal nerves. Consequently, in every case of acute abdominal pain the lungs, both front and back, should be carefully examined. The temperature in these cases is usually higher than in appendicitis, and the pulse-respiration ratio may be disturbed.

The pain of severe dysmenorrhœa, and of lead colic, and that met with in purpuric con-

ditions, may also give rise to diagnostic difficulties.

Treatment.—The trend of present surgical opinion is in favour of immediate surgical treatment in every case of acute appendicitis. Some surgeons even advocate operation in mild and doubtful cases, on the ground that the very vagueness of the symptoms constitutes an added danger. In support of their contention they point to the fact that the mortality from immediate operation is much less than in cases not operated upon. The physician, however, may be placed in a position where surgical assistance is only obtained with difficulty. It will then be necessary to differentiate between mild and severe cases; and in spite of the difficulties that have been so often referred to in judging the state of an appendix from the symptoms, it will usually be possible for a physician of experience to decide when it is safe to wait, and to rely on medical measures only.

Medical treatment, indeed, consists largely in careful watching. The patient must be kept in bed; diet is to be restricted to fluids, such as diluted milk, peptonized milk, albumen water, chicken tea, lemonade, and the like; hot applications may be employed over the appendix region to relieve the pain, but care must be taken not to injure the skin, in case an operation should become necessary; the bowels should be opened by means of a plain soap-and-water enema given slowly through a long tube and funnel, and repeated daily; and, as a rule, no medicine whatsoever should be given by the mouth. If severe vomiting, abdominal distension, or other acute symptoms call for treatment, the relief should consist of a surgical operation.

In mild cases two questions regarding medical treatment arise—(1) the administration of opium, (2) the use of purgatives. Even when the diagnosis has been made, *opium* is to be used with the utmost caution. If the diagnosis is doubtful, opium must not be given. Many a patient has died as a result of the cloaking of symptoms by this drug. Pain, if severe, can generally be relieved—at any rate in part—by aspirin and by local applications. If it persists it should be regarded as an indication for operation, and not for opium. *Purgatives* are an undoubted danger, and it is a safe rule to regard inability to secure an evacuation of the bowels by an enema, repeated on three or four occasions, as an indication for operation. As the symptoms subside, mild purgatives may

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be given, such as a dose of castor oil or 2 gr. of calomel, but while acute symptoms persist it is much safer to avoid all purgatives.

The physician should remember that operation, if performed at all in the acute stage, is followed by better results if done within the first twenty-four hours. If a surgeon is available, he should in all cases be asked to see the case at once, in order that a joint decision may be arrived at regarding the safety of delay. In severe cases surgical treatment should be obtained at all costs immediately.

If during the progress of a case that is being medically treated symptoms should point to the formation of an abscess, operation should be at once advocated. It sometimes happens, however, that a case is seen for the first time three or four days after the commencement of symptoms and at a stage when it is impossible to say whether an abscess is present or not. As operation in these circumstances is attended by less favourable results than when performed either immediately or after all symptoms have disappeared, it will be important to decide by a careful consideration of the history and of the physical signs whether the process is resolving or not. The existence of leucocytosis may here be regarded as pointing to the advisability of operating, and it must also be remembered that the persistence, after three or four days, of even a moderate rise of temperature is always suspicious. In cases of doubt it is wiser to operate at once. Delay, in order that a suspected abscess may become circumscribed, may result either in rupture and spreading peritonitis, or in the development of severe toxæmia and intestinal ileus. Even in abscess cases it is advisable, if possible, to remove the appendix itself.

"Interval cases."—If a patient has once suffered from appendicitis he is extremely liable to recurrence. Probably at least one-half of all patients suffer from one or more recurrent attacks. Consequently, the question often arises as to the course to be advised to a patient who has experienced an undoubted attack. As the mortality from operation during quiescence of inflammation is practically nil, operation should invariably be advised, unless the general health of the patient is such as to preclude any operative measure. In taking this course the physician should first satisfy himself that the patient has really suffered from acute appendicitis. Of late the appendix has been removed from many patients on the merest suspicion, and the removal has fre-

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quently been unattended by relief from the symptoms of which they have complained. If the diagnosis is certain, however, there is no doubt that the safe course for the patient to pursue is to submit to operation.

Chronic appendicitis.—It is in chronic appendicitis that most doubt is likely to arise regarding diagnosis and, in consequence, regarding treatment. Movable kidney, renal colic, gall-stones, chronic ovaritis, constipation, neurosis, and many other conditions may account for pain in the right iliac fossa. When, however, after exhaustive examination a definite diagnosis of chronic appendicitis is arrived at, removal of the appendix should be advised. If this be refused, careful regulation of diet, avoiding all indigestible articles of food, mild purgation, the avoidance of active exertion, and careful protection against chills should be advised. Any septic process in the body, such as bad teeth or chronically enlarged tonsils, should be dealt with, but sooner or later in most cases the occurrence of an acute or subacute attack will emphasize the need for a surgical operation.

T. G. MOORHEAD.

APRAXIA.—By the term apraxia or dyspraxia is meant inability to perform certain purposive movements voluntarily when there is no motor or sensory paralysis, or ataxia, to hinder or impair their performance. The patient may be, and indeed often is, able to use, in an automatic or involuntary fashion, the muscles concerned, thus showing the absence of paralysis, yet he fails to perform the given movement on request, in spite of his understanding what is required of him and endeavouring to carry it out. The condition has long been observed in hemiplegics with aphasia; they often are unable to put out their tongue at the word of command, but do it involuntarily when about to lick a stamp or take a spoonful of marmalade.

Apraxia may, however, be much more complicated. In such movements as striking a match to light a pipe the apraxic patient may become hopelessly "lost," and fumble with the objects in futile ways. Only certain types of movements may reveal the apraxia; others may be normally executed. Thus the patient may be apraxic for bilateral movements only, such as coughing, taking a deep breath, yawning, sniffing; individual movements of the extremities may be normal. Or the apraxia may concern only "reflexive" movements, such as putting the hand on the head, the finger

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on the nose, etc.; movements "away from" the patient may be correct. Further, apraxia may be unilateral or bilateral—a fact of the utmost significance. If it is confined to one side or one arm, it follows that the condition is localizable, just as is hemiplegia or Jacksonian epilepsy.

Evidence has been steadily accumulating which goes to show that there are two areas in connexion with lesions of which apraxia or dyspraxia is prone to occur. These are (1) the parietal region and (2) the frontal region and corpus callosum. It may be stated very briefly that the hypothesis is that there is a "centre" in the frontal lobe, in front of the motor area proper, where the series of consecutive muscular innervations requisite for the due performance of any given act are co-ordinated so that they occur in their proper sequence. This centre has been called the "eupractic centre;" it has been shown that Broca's speech centre and the so-called writing centre are in reality part of this wider eupractic centre. A lesion here will cause apraxia on the opposite side of the body. It is very probable that, owing to the lead of the left hemisphere over the right in right-handed individuals, a lesion on the left side may cause apraxia in the left limbs also. A lesion of the corpus callosum association-fibres from the left to the right hemisphere will produce the same effect. Further, a lesion which isolates or cuts off this eupractic centre from the main association-paths of the rest of the cortex—in particular, lesions in the parietal region cutting off association-fibres passing from the great parietal association-centres forwards to the frontal lobe—will also result in apraxia.

Apraxia, then, is not a disease, but merely a symptom, just as is aphasia. The prognosis and treatment of the symptom, therefore, are essentially those of the underlying pathological factor.

The commonest causes of apraxia are cerebral vascular lesions and tumours, and the reader is referred to the articles on **CEREBRAL VASCULAR DISEASE** and **CEREBRAL TUMOUR** for a consideration of this side of the subject. In addition, apraxia is often noted as a symptom in the course of such diseases as general paralysis of the insane, dementia præcox, senile and arteriopathic dementia, postepileptic confusional states, etc., and naturally the treatment is that of the disease concerned.

On the other hand, when apraxia is estab-

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lished in a stationary form, as a sequel to vascular or other lesions, treatment by re-education may well be considered. It will consist in the slow and painstaking re-training, for such acts as are lost or defective, of the limb or limbs concerned. Collier suggests that the regular taking out and replacing of the pegs of a cribbage board, or of the marbles of a solitaire board, would be useful exercises of a simple order. Should the apraxia be unilateral, the method of simultaneous employment of similar muscle groups of the two arms might be utilized with advantage. If the patient is apraxic for only a limited number of movements, re-education in this way is worthy of a trial.

S. A. KINNIER WILSON.

APROSEXIA (see ADENOIDS).

ARAN-DUOHENNE PARALYSIS (see MUSCULAR ATROPHY, PROGRESSIVE).

AROUS SENILIS (*syn.* Gerontoxon).—A greyish line around and slightly within the margin of the cornea. It is broadest above and below, and does not advance towards the centre. Although more common in elderly people, it may be seen in young adults or even in children. It is unaccompanied by symptoms. At one time it was thought to signify the presence of fatty degeneration of the heart, but this view is no longer held. It is caused by a peculiar form of fatty degeneration of the corneal cells and fibrillæ.

FREDERICK LANGMEAD.

ARGYLL - ROBERTSON PUPIL (see OPHTHALMOPLEGIA).

ARRHYTHMIA (see HEART-BEAT, ABNORMALITIES OF).

ARSENIO POISONING (see POISONS AND POISONING).

ARTERIAL DEGENERATION.—A local or general thickening of the arterial wall, with resulting diminution of the lumen, caused in atheroma and syphilitic arteritis by fibrous overgrowth in the tunica intima, and in diffuse arterio-sclerosis by hypertrophy of the muscle-fibres of the tunica media, usually with fibrous overgrowth in the tunica intima and in some cases in the adventitia.

Etiology. Wear and tear of life.—Atheroma and arterio-sclerosis are, generally speaking, diseases of middle or advanced age. As in the case of other organs, the health of the

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arteries is best served by preserving the correct balance between insufficient use and over-use; therefore one of the most important causes of their premature degeneration is an excessive wear and tear of life, entailing, as it does, undue strain on their walls. Hence the increased liability to arterial degeneration which exists under conditions of modern civilization. Under this heading comes arterial degeneration from excessive muscular exertion, severe emotional disturbances, or prolonged psychical stress.

Heredity.—The influence of heredity is well recognized. It is a common experience to meet with several members of one family who succumb at a comparatively early age to one or other of the results of arterio-sclerosis.

Chronic intoxications.—These include dietetic errors, typified by excess of food, especially of proteid food; constipation; imperfect digestion; and alcoholic excess. Immoderate smoking is another probable cause. Conditions of perverted metabolism, such as those associated with the rheumatic or arthritic diathesis, gout, chronic Bright's disease, obesity, and diabetes mellitus, are factors, as also is lead poisoning.

Infections.—Arterial degeneration may be a sequel of acute arteritis resulting from one of the acute infective fevers (*see* ENDARTERITIS, ACUTE). It may result from syphilis, one of the most potent causes, from enteric fever, and from tuberculosis.

High arterial blood-pressure, especially if continuous, is an important cause of arterio-sclerosis.

Pathology.—**Atheroma** or **endarteritis deformans** is a focal condition which affects the larger and medium-sized arteries. It is characterized by the presence of oval or circular raised patches on the inner coat, which are yellow and at first semi-transparent and gelatinous, but later become opaque, firm and dense. It must be distinguished from the patches of fatty degeneration which are met with in blood diseases, for these are not raised above the surface. In atheroma the elasticity of the vessel is diminished, and it may be dilated as well as elongated. The essential change occurs in that portion of the wall situated between the endothelium and the elastic lamina, and consists in a proliferation of the cells. Caseation, fatty degeneration, or calcification may occur in the patch. Microscopically, the atheromatous material which results from the softening of the patches consists of cholesterolin crystals, fatty acids, oil globules,

and disintegrating cells. The intima over the patch may yield, and a so-called "atheromatous ulcer" result. In the aorta the process occurs especially in the ascending part of the arch, and affects particularly the concave surface. It is most marked at the bifurcation and around the orifices of branches. The process may be much more advanced in some vessels than others. Primary calcareous infiltration of the media may occur as a process distinct from atheroma.

Diffuse or general arterio-sclerosis affects the arterio-capillary system generally, and is identical with the "arterio-capillary fibrosis" of Gull and Sutton. It begins in the arterioles and capillaries. The changes consist in great thickening of the wall of the artery with diminution in the size of the lumen. The length is increased, with resulting tortuosity; and the capillaries are also thickened. Microscopically, according to William Russell, the essential change is found to be a thickening of the media due to hypertrophy of the muscle-fibres. Later on, these may show hyaline swelling, fatty degeneration, or atrophic changes, by which the muscular elements are obscured. Besides this there is usually, but not always, a thickening of the intima (Russell) due to a hyperplasia of its subendothelial connective tissue, and in some cases fibrous overgrowth and thickening of the adventitia. The arterioles and capillaries may be completely obliterated. The process may affect the veins, the so-called "phlebo-sclerosis." The heart is hypertrophied, its walls being usually tough from an excess of fibrous tissue. There may be sclerotic changes in the valves, especially on the left side of the heart. The capsule of the kidneys is, in places, adherent and the organ firm, owing to an increase in fibrous tissue, but the most conspicuous feature is the thickening of the arteries.

Experimentally, pathological changes in the media and intima have been produced by inoculation with cultures of various micro-organisms, and it is especially interesting that injections of adrenalin and other drugs which raise the blood-pressure have been found to cause degenerative changes in the media.

Syphilitic arteritis affects especially the brain, aorta, and coronary arteries. Its most frequent seat is the brain, the arteries at its base, the vertebrals, the basilar and the carotids being particularly liable. It is a process distinct from atheroma. It may be limited to the aorta or the coronary arteries, but some-

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times the small arteries and arterioles generally are affected. As a rule, the limitation of the disease to the vessels of one organ or to a limited portion of one of the large arteries is characteristic of syphilis. The morbid appearances in the case of the aorta are described under AORTITIS, SYPHILITIC. In the cerebral or coronary arteries occur small, firm, greyish-white opaque nodules. As the growth proceeds the lumen may be almost obliterated. Microscopically there is a proliferation of the subendothelial layer owing to development of spindle and stellate cells and later on of fibrous material. In the large vessels there is also a periarteritis and mesarteritis. The nutrient arteries of the large vessels are much thickened, and there is extensive inflammation. Owing to this inflammation, rupture or dilatation, or the formation of an aneurysm at the diseased spot, may result. The proliferated subendothelial tissue shows little tendency to undergo either caseation or calcification. Hereditary syphilitic arteritis shows the same microscopic characters as the acquired disease.

Symptomatology.—Symptoms are protean, since they are chiefly the result of interference with the function of the organ or organs principally affected in each case. Again, they will vary to some extent according as the underlying pathological condition is general arterio-sclerosis, atheroma, or syphilitic arteritis. It may, perhaps, be stated broadly that general symptoms are, in a majority of cases, due to diffuse arterio-sclerosis, and symptoms referable to the brain and heart to syphilitic arteritis, while signs of impairment of function of a particular organ or part of the body may result from either general arterio-sclerosis or atheroma.

General symptoms.—In cases exhibiting high blood-pressure the earliest symptoms—which occur, in fact, before the sclerosis is established—are associated with the toxæmia that accompanies the high blood-pressure. This prodromal stage, called by Huchard that of “presclerosis,” is characterized by inaptitude for work, morning fatigue, headache, drowsiness, noises in the ears, and attacks of neuralgia or resembling migraine. These symptoms, if accompanied by high arterial blood-pressure and accentuation of the aortic second sound in a subject in whom one or more of the recognized causes have operated, would suggest early arterio-sclerosis. The condition resembles that described by Allbutt as “hyperpiesis,” in which, in persons beyond middle age, there

occur certain symptoms accompanied by a persistent rise of blood-pressure, but not associated with renal disease. The subject of arterio-sclerosis may appear prematurely old, perhaps somewhat ill nourished; the skin pale, sallow, or of an earthy appearance, dry, and somewhat inelastic; there may be arcus senilis. Among the most striking symptoms is a gradual loss of physical and mental vigour. In many cases, on the other hand, the patient is apparently in good health, well nourished, or even obese, his face of a high colour, and his cheeks showing venous stigmata.

Physical signs.—In those cases in which there is general involvement of the arterial tree, or in which the splanchnic arteries or thoracic aorta are extensively affected, the pulse shows the characteristics of high pressure, and the accessible vessels are thickened and perhaps tortuous, the lumen in some cases being smaller than normal, and in others, more advanced, notably larger. If the condition be general arterio-sclerosis, the thickening will be general and uniform, but if atheroma, the thickening will be localized and irregular, so that the wall may feel beaded to the finger. The “locomotive” pulse may be detected. The sphygmogram exhibits a sudden vertical percussion stroke, the summit is rounded or flattened, and the line of descent gradual, and, in general arterio-sclerosis, the dicrotic wave is in great part obliterated. In many cases, however, the superficial vessels show no obvious changes. Examination of the fundus oculi reveals irregularities in the calibre of the arteries—which are unusually tortuous—excessive brightness of the central light streak, and a kinked appearance of the veins which pass over these arteries. (PLATE 23, Fig. 6, Vol. II, facing p. 412.) With the signs of high pressure in the systemic arterial system are associated those of hypertrophy of the left ventricle of the heart—a heaving impulse, displacement downwards and to the left of the apex beat, and accentuation of the aortic second sound. In many cases there are signs pointing to dilatation of the arch of the aorta. Signs of arterial thickening or of high pressure may be completely absent if the disease is localized, and the pressure may in these circumstances be even below normal. If there is atheroma of the aorta, a systolic murmur may be heard in the second right intercostal space.

Local symptoms.—An important group of symptoms is that which is probably explained by the temporary impairment of function of

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a particular area due to the cutting off of its blood supply. These phenomena appear to be caused by a spasm of the muscular coat and consequent obliteration of the lumen of the artery which supplies that area, particularly if this artery be an end artery. Such spasm is, as William Russell has pointed out, especially liable to affect sclerotic arteries. Thus is explained the condition known as *intermittent claudication* (*angina cruris*). After walking, one or both of the lower limbs become affected by painful feebleness. If the patient continues walking the pain becomes almost unbearable, causing him to slacken pace and finally to stop completely. If examined, the limb is found to be cold and bloodless, and the pulse in the posterior tibial or dorsalis pedis arteries may have disappeared. The symptoms subside with rest, but the intervals between the attacks become shorter as time goes on. Gangrene may eventually supervene. When this comes on the pains are severe and exacerbated by movement, the skin of the toes and foot is cold, at first pale, then exhibiting patches of cyanosis, and finally becoming black. Although the lower limbs are usually affected the upper limbs are not exempt. In cases where the condition falls short of intermittent claudication, numbness, tingling, burning and shooting pains, or disagreeable sensations may occur in the limbs.

The pain may be situated in the calves, soles, or around the nails, and there may be attacks of painful cramp, especially at night, and coldness and pallor of the extremities. Or there may be merely a relaxation or giving way of the limb for a few seconds, and no pain.

In arterio-sclerosis of the cerebral vessels, transient hemiplegia or monoplegia, aphasia and paraplegia are not very uncommon. The attacks are sudden, and recovery is complete. The paralysis is rarely absolute. French neurologists have described a syndrome due to intermittent spasm of the arteries of the spinal cord, which resembles that of intermittent claudication. The symptoms are said to differ from those in the latter condition in that arterial pulsation persists in the feet, vasomotor phenomena are wanting, the deep reflexes are exaggerated, extensor response of the toes is present, and finally there is a frequent desire to micturate, as well as genital symptoms. The arteritis causing these spinal-cord symptoms is said to be almost always syphilitic. It is stated further that if the intermittent spasm affects the arteries of the

posterior columns it gives rise to symptoms resembling tabes. A similar cause has been invoked to explain certain abdominal symptoms, some of which have been associated with abdominal aortitis or sclerosis of the mesenteric or other abdominal vessels. Such are paroxysms of epigastric or umbilical pain, meteorism, distension, flatulence, vomiting, constipation, or even hæmorrhage from the bowel. They are often increased by physical or mental stress, but not usually by food.

Nervous system.—There may be heaviness and oppression, or headache which is apt to be frontal in situation and continuous or may resemble migraine. Other symptoms are irritability of temper, apathy, emotionalism, lack of power of concentration, defective memory, and drowsiness. The patient may suffer from sleeplessness or may find his sleep less refreshing than formerly. He may, in fact, exhibit the clinical features of neurasthenia. A gradual failure of mental powers is common. He loses his natural keenness in his affairs, and is less capable of their administration. His expression is dull. Finally, a state of dementia may occur, which may be associated with periods of excitement and mental vagaries. Mott states that in some patients there may occur attacks of maniacal excitement or depression with hallucinations and delusions, so that the clinical picture may resemble that of general paralysis of the insane, but that, as a rule, the dementia is not so pronounced as in the latter disease. A common symptom is vertigo. It often occurs on exertion, or follows a sudden change of posture, especially that from the recumbent to the upright position. It may accompany the crises of hypertension. The vertigo may be associated with tinnitus, but is not so severe as that which occurs in Menière's disease. Again, it may be accompanied by a slow pulse and syncopal or epileptiform attacks (Stokes-Adams syndrome). Epileptiform convulsions may occur in association with high blood-pressure. Osler says that, in the absence of syphilis and lead-poisoning, convulsions occurring in the middle-aged should always excite suspicion of arterio-sclerosis. In cases in which the brain arteries are involved cerebral hæmorrhage or thrombosis may ensue, the former being especially common as a cause of death. There may also be a cerebral embolism as the result of detachment of clot from an atheromatous aorta. If the pathological condition be a syphilitic arteritis there are certain characteristic symptoms and signs. They are the

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either to partial or complete obliteration of the arteries or to their dilatation. Under the first heading come the various pareses and paralyses which may occur; under the second, cerebral hæmorrhage, though this is less common in these cases than thrombosis.

Of paralyses, there is frequently a combination of a cranial-nerve paralysis with a hemiplegia; ocular palsies and aphasia are common. The transitory character of the early paretic and paralytic phenomena is especially important. Loss of motor power is much commoner than loss of sensibility, though various sensory phenomena may occur. There may also be various psychical disturbances, such as slow cerebration and loss of decision, and also fainting fits and losses of consciousness.

Circulatory system.—Failure of the hypertrophied heart to respond to the ever-increasing demands made upon it owing to the increased resistance to the circulation is a serious event, and is one of the common causes of death in high-pressure cases. It is for symptoms of the various stages of cardiac dilatation and failure that the patients most frequently seek treatment. In a typical case the impulse, while still forcible, is diffuse; a systolic murmur, the result of relative mitral incompetence from dilatation of the left auriculo-ventricular orifice, may be audible; there are râles at the pulmonary bases, and signs of failure of the right side of the heart, such as anasarca of the dependent parts of the body, and perhaps venous pulsation in the neck. Often, however, the pulse remains regular in force and frequency, and the blood-pressure readings may keep at their high level and be associated with accentuation of the aortic second sound. Dyspnoea on exertion, cardiac oppression or palpitation occur, and, indeed, these symptoms are common before there are any more serious signs of heart failure. Such a condition may yield satisfactorily to treatment directed towards diminishing the work of the heart, and attacks of this kind may occur many times before they lead to a fatal issue. Other cardiac signs met with are bradycardia or paroxysmal tachycardia, arrhythmia, and fainting fits. Narrowing of the orifices of the coronary arteries from arterio-sclerosis is a frequent cause of myocardial degeneration and weakness, the resulting symptoms being præcordial oppression, faintness, and dyspnoea. Sansom states that in a patient who is past the prime of life (or younger if he has had syphilis) who exhibits

dyspnoea on exertion, and whose heart and vessels are normal on examination, one should consider the possibility of atheroma of the coronary arteries. If a main branch is affected sudden death from thrombosis may result, and this is sometimes the first manifestation of arterio-sclerosis. Occlusion of the trunk of the coronary artery is sometimes the only lesion found in cases of sudden death. The involvement of the coronary arteries may be a cause of fibrosis of the heart, or the myocardial degeneration may occasionally lead to aneurysm of the heart or to rupture. Angina pectoris is not uncommon. In coronary obstruction due to syphilitic disease attacks of angina pectoris are frequent. With regard to the blood, although the patient exhibits pallor there may be no actual diminution in the number of red blood-corpuscles.

Urinary system.—Renal symptoms are characteristic of *general* arterio-sclerosis. They fall into two classes, those associated (a) with a granular contracted kidney, (b) with an arterio-sclerotic kidney. In the first the amount of urine passed is large, and the patient has to rise at night to micturate; the specific gravity is very low, and there may be a small amount of albumin with hyalin casts and often red blood-corpuscles. In the second there may at first be no albumin, and the specific gravity is normal or sometimes high; later the amount of albumin may be large.

Respiratory system.—Many cases are associated with the signs and symptoms of chronic bronchitis and emphysema of the lungs.

Digestive system.—There may be dyspeptic symptoms. In angina pectoris marked abdominal symptoms may occur—the so-called angina abdominalis. Ulceration has been found associated with disease of the vessels of the stomach and intestine.

Diagnosis.—In a typical instance of general arterio-sclerosis the physical signs and symptoms point at once to the diagnosis. A correct diagnosis, however, is of greatest importance in cases of "presclerosis," when it is not too late for judicious treatment to prevent organic changes. It is then that the sphygmomanometer has one of its chief uses. The cases most liable to be overlooked are those in which the superficial arteries are healthy and in which the patient either feels perfectly well or presents himself for some of the manifold symptoms of neurasthenia. If in such a case the blood-pressure is found to be raised and does not yield to treatment, and if at the same time

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signs of renal disease are absent, the diagnosis may be made with confidence. It might be clinched by an examination of the retinal arteries. A common clinical picture is that of a well-nourished middle-aged patient with a high colour and exhibiting venous stigmata on the face, who presents himself for shortness of breath and cardiac discomfort (both brought out on exertion), palpitation, and perhaps dyspeptic symptoms, and who, on examination, is found to have high blood-pressure with or without thickening of the superficial arteries, and an accentuated aortic second sound, together with râles at the base of one or both lungs.

In cases of localized disease, in the absence of changes in the superficial arteries, or of a clue given by ophthalmoscopic examination, or by signs of disease of the thoracic aorta, the diagnosis will depend upon the correct interpretation of the symptoms. In this connexion it must be remembered that the first symptoms of disease may be those of a fatal seizure, either cardiac or cerebral. Permanent thickening of the accessible vessels has to be distinguished from apparent thickening due to spasm of the muscular coat. In the former case the vessel may be smaller or larger than normal; in the latter it will be smaller, and the condition will disappear as the result of treatment. In an advanced case with resulting heart failure there may be difficulty in distinguishing it from one of primary valvular disease, particularly if, as not infrequently happens, a mitral regurgitant murmur be present. In such a case the examination of the pulse and arteries, not omitting that by an ophthalmoscope, perhaps the presence in the urine of hyalin casts, and a consideration of the history of the case, should enable the practitioner to avoid error.

Prognosis.—In diffuse arterio-sclerosis, if the disease be once definitely established the prognosis is bad. In atheroma the prognosis depends essentially upon the locality affected. If this be the brain or the heart, the outlook is very serious.

Treatment.—A sharp line must be drawn between cases of "presclerosis" and those in which permanent disease is established. In the former we can, by attention to general hygiene and by judicious treatment directed towards elimination by the means detailed below, prevent the development of anatomical changes; in the latter all we can hope for is to delay the progress of the disease and to treat complications and symptoms.

The patient should, above all things, avoid a strenuous life, either mental or physical, but both mind and body should be exercised, care being taken to stop short of fatigue. Sudden physical exertion, and as far as possible worry and anxiety, should be avoided. If active exercise is contraindicated, massage to the muscles of the limbs and thorax may be substituted. Meals should be small in bulk, particularly the evening meal, and limited in regard to the proteid constituents. Meat extracts should be avoided. A milk diet may be usefully substituted for meat once a week or for a few days every month. Roget and Gouget recommend such a diet in cases presenting symptoms of auto-intoxication. Tea and coffee must be limited; water may be drunk freely between meals. It should not be hard, but if a mineral water be preferred it is important to choose one containing only small quantities of saline constituents. Alcohol may be taken only in the strictest moderation; good whisky or Moselle is the best form. The most suitable climate is one which, while warm and equable, is also dry. Generally speaking, seaside resorts and those above an elevation of 2,000 ft. are unsuitable. A daily warm or tepid bath should be taken, but cold or very hot baths are contraindicated. The clothing must be warm. A free daily action of the bowels must be ensured, if necessary by means of saline aperients; a frequent liquid evacuation by means of a calomel purge is desirable.

In the case of high pressure associated with actual anatomical changes the amount of reduction, if any, which can be carried out with advantage to the patient can only be determined in any particular case by observation of the effect of carefully applied therapeutic agents upon the general condition and special symptoms. Venesection is a useful procedure in many cases.

The most generally useful drugs are the iodine compounds. Potassium or sodium iodide may be given in doses of 5-15 gr., thrice daily, for considerable periods of time; occasional intermissions being desirable in the case of the potassium salt. If the inorganic iodides are not well borne the organic compounds should be tried. Of these sajodin, in which the amount of iodine is one-third less than in iodide of potassium, and which may be conveniently administered in tabloids of 7½ gr., or iodipin, an emulsion of 1 dr. of which corresponds to 6 gr. of iodide of potassium, may be substituted. Guipaine, a drug obtained from

ARTERIAL DEGENERATION

mistletoe, is worth trying, and can be administered in pill form in 5-cg. doses, in amounts varying from 6-25 pills in the twenty-four hours. The hippurates or benzoates of ammonium are recommended by Oliver for the reduction of high pressure, especially if combined with ammonium bromide. These are safe remedies. The dose of the hippurate is 5-10 gr., and of the benzoate 5-15 gr. Thyroid extract is useful sometimes, especially in cases associated with obesity.

The vaso-dilator group of drugs is, generally speaking, best reserved for the relief of special symptoms. Erythrol tetranitrate has an action as prolonged as that of any other member of the group; it may be given in tabloid form in doses of $\frac{1}{2}$ or $\frac{1}{4}$ gr. Among the special conditions calling for treatment are those of vascular spasm in various areas, particularly the brain. In cases of spasm of the cerebral arteries with resulting paresis, treatment by vaso-dilators, especially erythrol tetranitrate, is indicated. Whether the use of this drug should be combined with stimulant or depressant treatment will depend upon whether the blood-pressure is low or high, and to determine this the blood-pressure instrument must be supplemented by the intelligent use of the finger. This may be exemplified by the case of a patient who has a cerebral seizure suggesting spasm. Perhaps the instrument will give a high reading, in part due to resistance of the arterial wall at the site of compression, although the actual blood pressure is not above normal. If, in obedience to the reading, lowering treatment be adopted, the result may be the development of cerebral thrombosis, whereas the finger, if used intelligently, may enable the practitioner to learn that the actual pressure is not high although the wall is greatly thickened, and that stimulant treatment is required.

In syphilitic arteritis the internal administration of potassium iodide should be combined with mercurial inunctions. Large doses should be given, and the treatment should be both early and prolonged.

OLIVER K. WILLIAMSON.

ARTERIES, SYPHILIS OF (see **ARTERIAL DEGENERATION**).

ARTERIO-SCLEROSIS (see **ARTERIAL DEGENERATION**; **CEREBRAL ARTERIO-SCLEROSIS**).

ARTERIO-SCLEROTIC KIDNEY (see **NEPHRITIS**).

ARTHRITIS, ACUTE INFECTIVE

ARTERIO-VEINOS ANEURYSM (see **ANEURYSM, ARTERIO-VEINOS**).

ARTHRITIC PURPURA (see **PURPURA**).

ARTHRITIS, ACUTE INFECTIVE.—This form of arthritis results from the entrance into a joint of bacteria, which reach it either through a wound, from the blood-stream, or by direct spread from a neighbouring focus of inflammation.

The infection may cause suppuration (see **ARTHRITIS, SUPPURATIVE**), or it may not progress beyond a non-suppurative inflammation. The latter form is seen (1) as the result of a penetrating wound, where resistance is good and only a few organisms have gained access to the joint; (2) when inflammation of neighbouring bone, bursa, glands, or cellular tissue causes an acute synovitis, which may subside as the primary focus gets better; (3) in certain general diseases, especially rheumatic fever, scarlet fever, gonorrhoea, syphilis, pneumonia, and dysentery. The last group is not considered in this article.

Symptomatology.—There is slight fever, and severe pain in the affected joint is complained of. The joint is distended with fluid, it is kept rigid in its position of greatest capacity, and the skin over it is red and hot.

Prognosis.—The outlook is in most cases good if from the beginning absolute rest of the joint is ensured, but some degree of prolonged or even permanent limitation of movement frequently results.

Treatment.—The limb must carefully be immobilized by splinting, after aspiration of the synovial effusion, if this can be done without penetrating suppurating tissues. Hot fomentations should be applied in the most acute stage; in less acute cases Bier's treatment by hyperæmia is efficacious. Small doses of a vaccine prep. from the infecting organism may be used. When the inflammation has quieted down, active movements, massage of muscles, and passive movements must be employed to restore mobility. Later on it may be necessary by cautious manipulations to break down adhesions under an anæsthetic.

C. W. GORDON BRYAN.

ARTHRITIS DEFORMANS (see **OSTEO-ARTHRITIS**).

ARTHRITIS, GONORRHOEAL (see **GONORRHOEA**).

ARTHRITIS, PNEUMOCOCCAL

ARTHRITIS, PNEUMOCOCCAL.—Pneumococcal arthritis is most common in young children and infants. The infection is conveyed to the joint by the blood-stream, and often causes a "primary" arthritis, no other focus of inflammation being apparent. The pharynx is usually the site of entrance of the organisms. There is sometimes a history of slight injury, and the arthritis may occur in the later stages of one of the exanthemata, especially scarlet fever, measles, diphtheria, and influenza. A pulmonary focus may precede its onset or may occur as a late event.

In adults "secondary" pneumococcal arthritis occasionally occurs about the ninth day of a definite lobar pneumonia.

Pathology.—One of the larger joints is usually affected, most frequently the knee, ankle, or shoulder. In mild cases the inflammation is confined to the synovial membrane, but in the more severe type the perisynovial tissues are infected and erosion of cartilage and cancellous osteitis occur. In most cases the joint fills with thick pus, sometimes greenish in colour, and there is a deposit of lymph in its interstices; occasionally, however, the effusion is serous, with flakes of lymph.

Symptomatology.—Usually the general reaction is slight, but sometimes the onset is accompanied by a rigor and there are high fever, toxæmia, and delirium. The local manifestations vary: in one form there is synovial effusion with slight increase of heat, very little limitation of movement and tenderness, and no pain; in other cases all the signs of suppurative arthritis are present—redness, oedema, great heat, tenderness, rigidity, with severe pain on movement.

Clinical varieties.—1. The commonest form is the so-called primary pneumococcal arthritis of young children, in which there is a rapid effusion into joint with slight local heat and redness, malaise, and fever, the temperature being about 100° F. Pain is not a salient feature. The joint-effusion is considerable, but the disease is far less severe in its signs and symptoms than streptococcal arthritis.

2. Occasionally, in older children, the arthritis takes a chronic course and closely simulates tuberculosis, from which, however, it is to be distinguished by the absence of severe pain, of rigidity, and of marked wasting.

3. A septicæmic form is sometimes seen in infants and is characterized by intense toxæmia, and usually accompanied by empyema and general peritonitis.

ARTHRITIS, SUPPURATIVE

4. In adults primary pneumococcal arthritis occurs but rarely; local and general symptoms are severe; the disease commences with a rigor and is indistinguishable clinically from other forms of suppurative arthritis.

5. In the secondary form following pneumonia the joint suddenly fills with pus; there is little local reaction, though general toxæmia is severe.

Prognosis.—In the first two varieties the outlook is good, and recovery with a movable joint occurs in most cases. In the others there is great danger to life, and, if recovery does occur, ankylosis is to be expected.

Treatment.—Aspiration through a wide-bore needle, repeated as soon as the joint refills, is the method of choice for the removal of the pus. If improvement in the general and local condition fails to ensue and the joint refills rapidly, as occasionally happens, an incision should be made under rigid aseptic precautions, and the joint washed out with saline and sewn up without drainage; subsequent aspiration may be necessary.

The joint must be rigidly immobilized by a splint which allows access without disturbance, the knee by a Thomas splint, the shoulder by a Middelдорpf triangle or a Jones abduction splint.

In the later stages the position of the joint should be varied from day to day, and active movements encouraged as soon as they can be performed without pain. Passive movements and massage are only suitable when the joint-condition is quiet and they cause no local or general reaction.

C. W. GORDON BRYAN.

ARTHRITIS, RHEUMATIC (*see* RHEUMATISM, ACUTE).

ARTHRITIS, RHEUMATOID (*see* RHEUMATOID ARTHRITIS).

ARTHRITIS, SUPPURATIVE.—Acute arthritis due to infection with pyogenic bacteria, there being acute inflammation of synovial membrane, ligaments, cartilages, and bone.

Etiology.—1. The causative organisms may gain access from without, through a wound due to operation, accident, or gunshot injury.

2. They may enter the joint from the blood-stream, in cases of pyæmia and septicæmia, and in acute diseases, particularly enteric fever, pneumonia, scarlet fever, and gonorrhœa. In "primary" cases there is no sign of

ARTHRITIS, SUPPURATIVE

acute disease elsewhere, the infection usually reaching the blood from the tonsil. There is often a history of strain or slight injury of the joint.

3. A joint may be infected by direct spread from a neighbouring focus, particularly the joints of the hand and fingers from a subcutaneous or tendon-sheath suppuration; the knee from prepatellar bursitis, and the elbow from olecranon bursitis; the knee, again, from a popliteal gland abscess, and the shoulder from an axillary adenitis or cellulitis; and any joint from a bone abscess, osteo-myelitis, or cancellous osteitis.

The causative organisms are commonly the streptococcus, staphylococcus, gonococcus, pneumococcus, and *B. typhosus*.

Pathology.—The entrance of bacteria produces an acute synovitis; suppuration soon follows, and, unless dealt with by prompt measures, the infection spreads outside the joint, and either reaches the skin or tracks along intermuscular planes or tendon-sheaths. The synovial membrane, at first swollen and hyperæmic, is converted into granulation tissue; the ligaments become œdematous and stretched, and pathological dislocation may occur early. The muscles are kept rigid, and undergo rapid atrophy and fatty degeneration. The cartilages are eroded, and exposure of the cancellous bone is followed by osteitis; the bone is rarefied and its vessels thrombose, leading to necrosis, and often to pyæmia; the periosteum is inflamed, and may be stripped up by a subperiosteal abscess.

Symptomatology.—The general symptoms are those of acute toxæmia with high temperature and rapid pulse. An increasing pulse-rate is of grave import and calls for radical treatment if the patient's life is to be saved. In severe cases the temperature is often not more than 100° F. and affords little guidance.

Great pain, leading to exhaustion from lack of sleep, is complained of, and the joint, which is held rigid, is tender, hot, red, and swollen; it is distended with pus and the skin is œdematous. Later, the suppuration extends outside the joint and spreads along intermuscular planes or reaches the subcutaneous tissue; in a wasted limb a large intermuscular abscess may easily be overlooked.

When the cartilages are eroded, starting-pains occur, from the rubbing together of the acutely inflamed ends of the bones. Any movement or jolting causes extreme pain with sudden muscular spasm. The joint is held in

the position in which its capacity is greatest, usually one of semiflexion. The ligaments being sodden and stretched, abnormal mobility becomes evident, and in the hip or knee pathological dislocation may follow. The onset of pyæmia is indicated by rigors; these are a grave sign, and are followed by the formation of pyæmic abscesses in other parts of the body.

Diagnosis.—In all suspected cases there must be no hesitation in inserting an exploring needle. In children it is a safe rule to regard every case of acute monarticular arthritis, accompanied by fever, as suppurative until it is shown to be otherwise.

Prognosis.—In all cases there is grave risk to life, and treatment must be prompt. Adequate early treatment may be followed by recovery, even with some degree of movement of the joint, though ankylosis is the common result. The prognosis in joints of the upper limb is much better than in the hip, ankle, and especially the knee, where suppuration frequently necessitates amputation and often causes death. In the acute stages death is due to toxæmia and exhaustion; the heart-muscle degenerates, the pulse-rate rises, and sudden syncope may supervene. Death is due sometimes to septic emboli being carried to the lungs or other vital organs. If the condition settles down to subacute suppuration with sinuses, the risk of lardaceous disease must be remembered; the onset of polyuria, albuminuria, or diarrhœa is serious, and in such a case amputation must be considered.

Treatment.—Throughout the active stages of the infection rest is essential; the patient must be kept in bed, and the joint fixed by a splint which ensures absolute immobility, in the position which will give the best functional use if ankylosis follows. The best type of splint is one which combines immobility with fixed extension, such as the Thomas knee-splint; a great advantage of such splints is that they allow access for examination and dressing without any disturbance. This is of the greatest importance and is too frequently overlooked; it is very harmful for the limb to be lifted from its splint each day, with consequent pain and auto-inoculation.

The joint must be emptied by aspiration or by drainage. In an early case the effusion is aspirated and preserved for bacteriological examination, a vaccine being prepared from the organism isolated. Following aspiration, the injection into the joint of various drugs such

ARTHRITIS, SUPPURATIVE

as formalin and ether has been tried, but any benefit from their use is doubtful, and in some cases they have certainly done harm; ether especially is liable to be followed by leaking and a virulent sloughing intermuscular cellulitis. Simple aspiration is safe and equally beneficial; if the joint is emptied early and the infection is not virulent, improvement in the general and local condition is noted next day; the joint will generally have refilled, but it is less painful and tender, and the temperature and pulse-rate are lower. In these circumstances, aspiration is repeated daily, and the character of the fluid observed; if it becomes gradually less purulent, recovery without drainage will result. If, however, the result of the first aspiration is the withdrawal of thick pus, or if the fluid is more and more purulent each time, or if the general condition does not improve, the joint must be drained.

In draining a joint, considerations of gravity, accessibility, and surrounding structures are all-important. As far as possible stiff rubber tubes should be avoided: they do not provide the best drainage, and may cause erosion of cartilage. In many cases free incision suffices, without the introduction of any form of drain; a dressing of gauze soaked in liquid paraffin containing 1 per cent. iodoform is the best. Where, however, the joint is not near the surface and the wound would become closed by overlying muscles, the best form of drain is a sheet of thin rubber (smooth or corrugated) introduced into the joint along the whole length of the incision. The limb must be arranged so that the incision is at a dependent spot.

If simple drainage is not followed by rapid improvement, a Carrel tube should be introduced into each recess of the joint, eusol being run into each tube two-hourly, day and night. The tubes are brought through the dressings, and it is convenient to join them up to one larger tube connected to a flask of the solution hung above the bed, the flow through this tube being controlled by a stopcock.

All extra-articular abscesses must be counter-drained by large tubes.

If, in spite of these measures, toxæmia increases and the pulse-rate rises, amputation must not be delayed. This grave step is rarely called for in arthritis of the upper extremity, but it must often be resorted to early in arthritis of the knee or ankle, as the only means of saving the patient's life. Risk from delay is unjustifiable, especially as severe suppuration means a long and painful illness, with a

partly crippled limb at the end, even if eventual recovery ensues; delay may allow the patient to pass the stage when he is fit to bear amputation; and modern prosthetic apparatus for the lower limb is comfortable and satisfactory in nearly every case. In chronic suppuration the onset of lardaceous disease is an indication for amputation.

The amputation wound should be left open, for closure later by extension and secondary suture. Gas-oxygen anaesthesia, or a local method (intrathecal novocain is the most generally useful), should be used in toxæmic cases.

When amputation is avoidable, excision is advisable in certain circumstances, particularly for pathological dislocation, for ankylosis in a useless position, and when cancellous osteitis is a prominent feature.

SPECIAL JOINTS

The **shoulder** is most commonly infected from axillary cellulitis or a penetrating wound; abscesses may point in front of or behind the deltoid, or in the axilla. Drainage is necessary in most cases, by incisions in front of and behind the deltoid, rubber-tissue being introduced along the whole length of each wound to keep its edges apart. Carrel's treatment is unsuitable. The best splint is one described by Sir Robert Jones, fixing the arm in abduction to 50° with slight flexion, the palm being directed towards the face. A Middeldorpf triangle is convenient, but does not give so good a position for ankylosis. Where there is an axillary wound, however, extension is applied, and the limb, abducted to 50°, flexed and supinated, is suspended from a Balkan frame. In the later stages suppuration often occurs under the supraspinatus fascia and necessitates counter-drainage. Prolonged sepsis, osteitis of the head of the humerus (shown by X-rays), and ankylosis in an adducted position are indications for excision, after which a movable joint usually results.

In the **elbow-joint**, drainage is obtained by incision on each side of the triceps tendon. A splint combining immobility with accessibility is Jones's skeleton elbow-splint. The joint is flexed just below a right angle, with the forearm in a position of partial supination. Excision is practised for faulty ankylosis, but should be postponed for several months after the inflammation has subsided.

The **wrist** is immobilized in extreme dorsiflexion on a skeleton metal splint, or it may be

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slung in this position on a Thomas arm-splint, with strapping extension applied to the hand and fingers. Great disability may result from matting of tendons, and active movements of the fingers should be encouraged as soon as the acute stage is over.

Suppuration in the **hip-joint** arises from osteitis of the head and neck of the femur, penetrating wounds, and pyæmia; it is especially common in enteric fever. Local symptoms are not as a rule acute, and the condition is easily overlooked; the joint becomes distended with pus, and a pathological dislocation may be the first thing that calls attention to the condition. In the early stages there are pain and rigidity, the joint being everted, with abduction masked by scoliosis, and flexion masked by lordosis. The head of the femur usually becomes necrosed and detached. Swelling is not obvious owing to the depth of the joint, and in all cases of suspected infection there should be no delay in exploring with a large-bore needle.

In the early stages drainage is established through an anterior incision to the inner side of the tensor fasciæ femoris; in late cases with profuse suppuration, counter-drainage in the buttock will be necessary. If the head of the femur is detached or extensively diseased it must be removed. The limb is fixed in full abduction, extension, and slight external rotation by weight-and-pulley extension, the pelvis being fixed by a Liston splint on the opposite side.

Suppuration of the **knee-joint** in adults is most commonly the result of a wound, but a blood-stream infection is frequent in children; it causes early and severe toxæmia with grave danger to life. In the early stages, and throughout the disease if careful treatment and absolute fixation are carried out, only the front of the joint is affected, its posterior part being shut off by the swollen ligamentum mucosum; to aid this the popliteal space should be supported by pressure. The subcrureus pouch is distended, the skin is hot, and there is œdema over the subcutaneous surface of the tibia. Pain is severe, and destruction of the articular cartilage takes place early. The joint is flexed, and pathological dislocation may follow, the tibia being flexed, externally rotated, and displaced outwards and backwards. Extra-articular abscesses form under the vastus externus and in the calf, and may easily be overlooked.

If the condition does not rapidly subside

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under treatment by aspiration, the joint must be drained. The best incision for draining an anterior arthritis is one above the patella, the limb being slung almost vertical, or the patient lying prone. For posterior arthritis counter-drainage through the popliteal space should be established. Carrel's treatment is applicable to this joint. Another method is to make a free incision on each side of the patella, cover the raw surfaces with gauze, and pack with sodium chloride tablets.

The knee is immobilized by a Thomas knee-splint with fixed extension by strapping, the leg and thigh being packed with wool to prevent lateral movement; the splint is suspended so that its ring is just clear of the bed when the limb is horizontal; but if the subcrureus pouch is being drained the foot must be raised until the limb is nearly vertical. Immobility and extension must be depended upon for the relief of pain, morphia being contraindicated where there is toxæmia.

Excision may be rendered necessary by cancellous osteitis. If the patient goes downhill in spite of careful conservative treatment, the thigh must be amputated before he reaches a state of serious toxæmia; hesitation in deciding to sacrifice such limbs has cost many lives.

The **ankle-joint** is not infrequently infected as a result of compound fractures in this region. Synovial effusion is apparent as a swelling in front of the joint and at each side of the tendo Achillis. An incision on each side is made for drainage, but in many cases adequate drainage can only be ensured by excision of the astragalus; the disease often commences as an osteitis of this bone, which is also an indication for excision. A Jones skeleton splint may be used, or a flexed Thomas knee-splint, in which the foot is suspended by a bandage glued to the sole. Throughout the disease the ankle must be kept at a right angle, with the foot in a slightly varus position. Ankylosis with adherent ligaments and tendons leads to considerable crippling, and in cases of severe toxæmia amputation through the middle of the leg should not be unduly postponed.

C. W. GORDON BRYAN.

ARTHRITIS, SYPHILITIC.—In **congenital syphilis** arthritis is not uncommon. In infants acute suppurative arthritis may be associated with acute epiphysitis; in older children a subacute synovitis occurs. The latter type usually makes its appearance between the

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ages of 6 and 12 as a unilateral synovitis with considerable effusion. There is little pain, and movements are free. The muscles are only wasted in late stages. The disease takes a chronic course and is very resistant to local treatment.

Diagnosis.—The possibility of congenital syphilis should be thought of, if in a case of suspected tuberculous joint the X-ray finding is negative. In tuberculosis pain is a marked symptom, there is little fluid, the synovial membrane is much thickened, and progress is more rapid. The diagnosis of syphilis is confirmed by family history, signs in other parts of the body, and a positive Wassermann reaction.

Treatment.—General treatment by mercury is of most importance, and, if it is commenced early, complete recovery is likely to result. Local treatment consists in aspiration and the application of Scott's dressing, with massage of the thigh.

Acquired syphilis.—In the secondary stage a symmetrical hydrops occurs, usually in the knees; the amount of fluid varies from day to day, and there is no pain. Scott's dressing is applied and the thigh is massaged; the arthritis gets well as the general disease gives way to treatment.

In the tertiary stage three types of arthritis are seen:

1. *Perisynovial gummatosis*, in which one or more gummata develop, most often in relation to the sterno-clavicular and knee-joints. Involvement of the skin is followed by a typical ulcer with clean-cut edges and sloughing base.

2. *Synovial gummatosis* is characterized by nodular thickening of the synovial membrane with effusion. The knee is most commonly affected. The disease resembles tuberculosis, but there are less pain and rigidity and more fluid, while muscular wasting is not a marked feature. Rigid splinting is unnecessary, but in the lower limb it is advisable to take the weight off the joint by a Thomas calliper splint. Early passive movements should be employed, as there is a great tendency to fibrous ankylosis. If neglected, the disease tends to progress to the type now to be described.

3. *Chondro-arthritis*.—The characteristic features are changes in the cartilages; their cells proliferate, the matrix is fibrillated, and erosion follows, with pitting of the ends of the bones. The symptoms are those of a chronic arthritis, with gradually increasing stiffness. The disease resembles osteo-arthritis. In syphilis, however, pain is absent, there is less crepitus, and the

ARTHRITIS, TUBERCULOUS

erosion does not correspond to the points of greatest pressure, while osteophytes and lipping are absent. Under general antisiphilic treatment the arthritis becomes quiescent, but ankylosis is a common result, so the joint should be splinted in the most useful position. Excision of the shoulder, elbow, or wrist may be necessary to restore function if fixation in a bad position occurs.

C. W. G. BRYAN.

ARTHRITIS, TUBERCULOUS.—This term is applied not only to cases of actual disease of a synovial cavity, but also to those in which there is tuberculous cancellous osteitis of the end of one of the bones which form a joint, though the actual synovial cavity is not infected.

Etiology.—The disease usually affects children, most cases occurring between the ages of 6 and 12, but it is not uncommon in young adults. As in tuberculosis elsewhere, general debility is a predisposing cause. The disease frequently follows a slight injury, but is uncommon as a sequela of severe injury of a joint. Probably the *Bacillus tuberculosis* usually enters the body by the tonsil, the alimentary canal, or the lungs, but the primary focus is not as a rule discoverable, the joint-disease being the only apparent lesion.

Pathology.—The infection either reaches the synovial membrane direct from the bloodstream or, more commonly, spreads from one of the bones of the joint. Occasionally it spreads from periosteum or an adjacent bursa. In children a bony origin is usual, the primary focus being in the diaphysis close to the epiphyseal line, and causing symptoms referred to the joint before the synovial cavity is actually invaded. In adults the disease begins in bone and in synovial membrane with about equal frequency; if in bone, the focus is situated under the articular cartilage and, by ulcerating through this, infects the joint.

The synovial membrane is at first thickened and hyperæmic, containing tubercles beneath its surface; caseous foci form and burst into the joint, the synovial membrane being converted into cedematous granulation tissue, with an external zone of fibrosis. The ligaments are swollen, and in the later stages the cartilages become eroded; the bone-ends are thus exposed in the articulation in a state of rarefying osteitis. Abscesses form, and if they reach the skin the resultant sinuses convey pyogenic bacteria to the joint.

Symptomatology.—The onset is usually

ARTHRITIS, TUBERCULOUS

gradual, but it is acute if a tuberculous abscess suddenly bursts into a joint; such sudden invasion is common in the hip, the disease commencing in the lower part of the neck of the femur close to the epiphyseal line, which is situated inside the joint-capsule. The symptoms may date from a slight injury.

Pain is a prominent symptom, aching in character, worse at night and after exercise, in the early stages relieved by rest. When the cartilages have become eroded, night starting-pains supervene. The characters of this symptom are very definite: while the patient is awake the joint is fixed by muscular spasm and the exposed ends of the bones are immobile, but as he falls asleep the muscles relax, the bone-ends rub against one another, sudden muscular spasm recurs, and the patient starts awake in agony, often with a scream.

An early sign is muscular rigidity, limiting all movements of the joint. The muscles of the limb are wasted, exaggerating the thickened appearance of the joint.

The joint is hot and swollen from thickening of the synovial membrane, and in occasional cases from synovial effusion.

Interference with function results from pain and rigidity, and in the lower limb a limp is often the earliest symptom. The limb is kept in a position characteristic for the particular joint diseased.

In late stages cold abscesses, giving fluctuation, approach the surface, and may burst, leaving sinuses lined by pale granulations.

Relaxation of ligaments and erosion of bones allow pathological dislocation to occur; in this stage examination under anaesthesia will show abnormal mobility.

Diagnosis.—The important diagnostic features are the rigidity in all directions and the early wasting. In all cases of suspected tubercle of bone taking part in a joint, X-rays should be used, and will show general rarefaction of the cancellous bone, a bone cavity, and in later stages irregularity of the articular surfaces. In the chronic arthritis of *congenital syphilis* there is little pain, and synovial effusion is a marked feature. *Synovial gummatosis* of tertiary syphilis may be distinguished by the absence of severe pain (and by signs of syphilis elsewhere. In *rheumatic arthritis* there is little synovial thickening, and usually several joints are successively affected. *Rheumatoid arthritis* and *osteo-arthritis* most commonly affect adults; in the former disease there is effusion, movements are free, and

thickened synovial fringes can be detected; in the latter, the pronounced bony changes with grating on movement are distinctive. In *infective and suppurative arthritis* the onset is acute, the course of the disease is rapid, and there is leucocytosis.

Prognosis.—In children, under careful treatment the disease may be expected to become quiescent if the limb is kept immobile for a long time and the general surroundings are favourable. A permanent cure often occurs at puberty. Ankylosis is the best result to be expected in most cases, though a movable joint is occasionally obtained.

In adults, especially after 40, the prognosis is bad; conservative treatment in the case of the knee and ankle should not be persisted in too long, and amputation will frequently be the only way of restoring the patient to activity.

Sinus-formation with prolonged suppuration may lead to lardaceous disease, and general miliary tuberculosis sometimes supervenes.

Treatment.—**General hygienic treatment** is of importance—fresh air, mental rest, and a liberal diet containing plenty of easily assimilable fats. Sources of chronic septic infection, such as hypertrophied tonsils, carious teeth, and constipation, must be looked for and treated. Use should be made of tuberculin, beginning with a dose of 1/100,000 mg., repeated once a fortnight in gradually increasing amounts.

Of local treatment the most important feature is absolute immobilization by the method of splinting best suited to the joint affected. Plaster of paris, celluloid, and the various splints devised by Thomas are of most general use.

Bier's passive congestion is sometimes helpful, the rubber bandage being applied firmly enough to produce a pink colour in the limb, without pain.

In the rare cases in which synovial effusion with melon-seed bodies is present, the joint is aspirated and washed out through the needle with 1·5-per-cent. saline solution. An emulsion composed of iodoform 10 parts, water 20 parts, and glycerin 70 parts may be injected and left in the joint. A cold abscess is treated in a similar way, but if it invades the skin it must be opened with rigid asepsis and cleared of its contents; after the application to its wall of BIPP (bismuth 1, iodoform 1, paraffin 2), it is sutured without drainage.

For sinuses, gauze soaked in liquid paraffin containing 1 per cent. iodoform is the best

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dressing; weekly injections of bismuth paste (bismuth subnitrate 6, calomel 1, olive oil 2, vaselin 12) sometimes promote healing.

Operative treatment is indicated in some cases. If the disease is localized as a small focus in bone, it is possible sometimes to eradicate it before the joint itself is infected.

Complete excision of the joint is not often called for, but a modified operation may be indicated—

(1) If the joint is disorganized and the bones are extensively diseased, with sinuses and secondary infection.

(2) To avoid ankylosis in a bad position, especially in the elbow and temporo-maxillary joints.

(3) In the shoulder, where the disease takes the form of a dry caries of the head of the humerus and synovial infection is a secondary feature.

The joint is opened, the diseased synovial membrane removed, carious bone eradicated, and the wound sutured, a rubber-tissue drain being left in position for forty-eight hours.

Amputation may be necessary—

(1) In elderly people.

(2) When the disease progresses in spite of careful conservative treatment, and the general health becomes much affected.

(3) When excision has failed.

(4) When extensive abscesses tracking down the limb cause disabling destruction of its soft parts.

(5) In certain cases of tuberculosis of two joints.

Careful treatment must in all cases be kept up for at least a year after all signs of active disease have disappeared, and for a much longer period a careful watch must be kept for recrudescence.

SPECIAL JOINTS

Tuberculosis of the **shoulder** is rare in children, but is fairly common in adults, commencing in the head of the humerus as a caries sicca. The limb is kept rigid in adduction, and wasting of the deltoid, the scapular muscles, and the arm is noticeable. Abscesses point in front, tracking down the tendon-sheath of the biceps, and occasionally behind the deltoid.

In children the joint should be fixed by plaster of paris, abducted, slightly flexed, and externally rotated; eradication by operation on the focus in the diaphysis is sometimes possible. In adults, cure by conservative methods takes a long time, whereas excision

gives good results, often with a movable joint; it is in most cases the best method of treatment.

Tuberculosis of the **elbow** occurs in older children and young adults. In children it commences usually in the superior radio-ulnar joint, spreading secondarily to the elbow-joint; in adults it begins as an osteitis of the olecranon or of the outer condyle.

There is at first loss of the movements of pronation and supination, with swelling and tenderness related to the head of the radius; later, swelling in front of and behind the elbow appears. An abscess may point on either side of the olecranon, or on the inner side of the arm tracking beside the ulnar nerve.

The joint should be fixed in lateral plaster splints flexed just below a right angle (i.e. at 70°), with the forearm midway between pronation and supination; with ankylosis in this position the patient will be able to dress, eat, and write. For ankylosis in a bad position an operation should be performed.

At the **wrist** the disease usually begins in the tendon-sheaths; there may be infection of the carpal bones or joints, or of the radius. It is the commonest joint to be affected in old people. Abscesses point either dorsally or in relation to the flexor carpi radialis.

The joint must be splinted in hyperextension by plaster-of-paris bandages; to obtain this position an anæsthetic may be necessary. When the disease has become quiescent, prolonged fixation by a skeleton hyperextension splint is continued; great disablement is the result if any other position is chosen.

Sacro-iliac disease is rare and usually occurs in adults. There are pain in the buttock and in the front and back of the thigh, and a sense of weakness increased by standing and, particularly, by any such exercise as involves contraction of the abdominal wall. The innominate bone on the side of the disease becomes tilted so as to give an appearance of lengthening of the corresponding limb. If the pelvis is supported the hip can be passively moved without pain; manipulations of the pelvis which compress or separate its two sides cause pain. In later stages there is tenderness on pressure posteriorly, and cold abscesses may appear in the region of the joint behind, laterally above the crest of the ilium, anteriorly above or below Poupart's ligament or in the ischio-rectal fossa. Ankylosis of the joint following tuberculosis may lead to interference with parturition.

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Diagnosis must be made from hip disease, from sciatica, from spinal caries, and from new-growth of the ilium. X-rays will show rarefaction of bone with irregularity of the joint-surfaces.

In conservative treatment the patient is kept recumbent, a plaster case being applied to the pelvis. If X-rays show localized bone-disease of the ilium, it may be eradicated by operation.

The **hip** is the most commonly affected joint in children, and its depth and liability to other infections and injuries make diagnosis an important matter. The disease usually begins as a cancellous osteitis of the lower part of the neck of the femur near its head. A focus of caries developing, in many cases the child is brought for treatment in this stage, with a limp and an aching pain. In others nothing is noticed until an acute arthritis results from the tuberculous focus bursting into the joint; after an acute onset the arthritis settles down to a chronic course.

As the disease progresses, three stages can be distinguished. In the *first* there are limping and pain, which may be localized in the hip but are often referred to the knee; pain in the knee in a child should be viewed with suspicion, and the hip-joint must always be examined. On inspection, wasting of the muscles of the thigh is seen; the earliest degree shows itself in the buttock by a lessening of the gluteal fold. The hip is flexed, abducted, and externally rotated; the flexion is usually masked by lordosis, which can be got rid of by fully flexing the sound hip, whereby the degree of flexion of the diseased one is made obvious. The abduction is masked by a lateral tilting of the pelvis, giving an appearance of lengthening of the limb on the side of the disease; after putting the two limbs in corresponding positions they are measured, and it is shown that there is no real difference in length. There is limitation of all movements, particularly those of hyperextension and rotation; in the earliest stage, when the disease is limited to the bone, rigidity may be shown only by a loss of hyperextension, tested for in the prone position.

In the *second* stage of the disease the cartilages are eroded, and night fever and starting-pains occur; the patient is unable to walk, and is inclined to lie on the sound side, the diseased hip taking up a position of flexion, adduction, and internal rotation, with an appearance of shortening; lordosis masks the flexion, and

scoliosis is present with a lumbar curve convex towards the sound side.

In the *third* stage, pathological dislocation has occurred from the laxity of the ligaments and destruction of the head of the femur and the margin of the acetabulum. Real shortening exists, and is estimated by measurement and by examining the relation of the great trochanter to Nélaton's line and by Bryant's triangle.

Abscess-formation may occur in the second and third stages; the abscess may track through the capsule in front and give rise to a fluctuant swelling to the inner side of the tensor fasciæ femoris, or it may leave the joint behind and appear in the buttock or in Scarpa's triangle; a large cold abscess may form under the fascia lata externally. Occasionally the osteitis spreads through the acetabulum, and a pelvic abscess results, appearing either in the ischio-rectal fossa or above Poupert's ligament.

Not only as a means of diagnosis, but to show the amount of bone disease, an X-ray examination must be made in all cases.

Diagnosis.—In doubtful cases examination by X-rays will usually distinguish tuberculosis from the conditions that simulate it, especially incomplete fractures of the neck of the femur and displacements of the epiphysis. A simple *traumatic synovitis* recovers completely during a fortnight's rest in bed. Limitation of all movements is the important sign of hip-joint tuberculosis that distinguishes it from *coxa vara*, from *congenital dislocation*, from *spinal caries* with psoas spasm, and from *sacro-iliac disease*. In older people *osteo-arthritis* may closely simulate tuberculosis; the presence of grating, thickening of the great trochanter, the X-ray appearances, and the alleviation of pain and stiffness by exercise are characteristic of that disease. In *suppurative arthritis* and *osteo-myelitis* there is high temperature with leucocytosis. In *syphilitic epiphysitis* there will usually be other signs of the general disease, and the Wassermann reaction is positive. In *osteo-chondritis juvenilis*, while flexion and extension are free, the other movements are limited. X-rays show alteration of the epiphyses for the head and both trochanters, with widening and irregularity of the epiphyseal lines.

Treatment.—At the beginning of treatment the patient must be kept in bed in every case, a weight extension being applied to exert traction in the line of the thigh. As the muscular spasm is overcome, the limb is

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brought gradually to a position of full abduction and extension; the bed must be made on a fracture board, the pelvic tilting must be corrected, a Liston splint being applied on the sound side, and lordosis must be abolished by increasing at first the flexion of the diseased hip. A plaster case is then applied enclosing the whole limb and extending up to the chest-wall, a window being left for observation of the postero-external aspect of the hip.

When the disease has become quiescent, in young children a double Thomas hip-splint may be used; after the age of 6 a single Thomas hip-splint with pelvic band may be fitted, and the patient is allowed to get about on crutches, with a patten on the foot of the sound side.

Fever, starting-pains, and abscess-formation are indications for absolute rest in bed, with traction as described.

Excision is occasionally indicated for extensive bone disease with secondary infection and toxæmia, or for threatening lardaceous disease. Amputation by the Furneaux-Jordan method must on rare occasions be resorted to.

In the **knee-joint** tuberculosis starts in synovial membrane and bone with about equal frequency; it is fairly common in adults. The joint is held rigid in a position of slight flexion, and there is much wasting of the thigh and leg muscles, which exaggerates the swelling of the joint. The edge of the thickened synovial membrane is palpable, and occasionally in the early stages there is definite synovial effusion. In later stages starting-pains occur; when disorganization is advanced, a quadruple displacement of the tibia supervenes, the leg being flexed, externally rotated, and displaced backwards and outwards.

The patient should be treated at first in bed. Flexion is overcome by the application of traction by strapping, the knee being supported on a pillow; by raising the foot of the bed the weight of the patient supplies the counter-extending force. The safest position for ankylosis is with the knee straight; when this position has been procured by traction the limb is encased in plaster of paris until the disease is quiescent. A Thomas knee-splint is then fitted, transmitting the whole of the patient's weight from the tuber ischii to the ground, and he is allowed to get about. At all stages care must be taken that foot-drop does not occur. After the disease is cured a celluloid or leather case reaching from the upper

third of the thigh to the lower third of the leg must be employed for a long time, to avoid the tendency to gradually increasing flexion.

In adults it is sometimes advisable to perform a modified excision operation if the bones are extensively diseased and there is secondary infection. After the age of 45 the prognosis is very bad, and at the best a cure could only be obtained after several years of total disablement during treatment; in the majority of such cases, therefore, amputation should be resorted to without undue delay.

In the **ankle-joint** the disease sometimes begins in adjacent tendon-sheaths; it may commence, however, in the astragalus or the tibia. Swelling is evident in front, and behind at each side of the tendo Achillis, and the calf is much wasted. The joint is kept in a position of plantar flexion, and the movements of flexion and extension are lost.

Treatment.—The joint is cased in plaster, the foot being at right angles to the leg in a varus position. The patient may get about on crutches, or with more convenience by the provision of a Thomas knee-splint.

For extensive osteitis, excision of the astragalus is sometimes indicated. In adults, especially, the prognosis is not good, and even after a cure of the tuberculosis the patient will suffer considerable disability from ankylosis and the fixation of tendons, with a constant tendency to attacks of fibrositis from strains and slight injuries. Unless, therefore, rapid improvement results from conservative treatment, a Syme amputation, or one at the middle of the leg, must be considered; with modern apparatus, these operations give extremely good functional results with practically no disability.

Disease of the **tarsal joints** usually begins in bone and spreads to the complicated synovial cavities. In children conservative treatment is carried out by the application of a plaster case, with the foot in a varus position; a useless foot is the common result. In adults the correct treatment, if the disease is extensive, is a Syme amputation.

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ARTHRITIS, VILLOUS (*see* RHEUMATOID ARTHRITIS).

ARTIFICIAL RESPIRATION (*see* RESPIRATION, ARTIFICIAL).

ASCARIS LUMBRICOIDES (*see* INTES-TINAL WORMS).

ASCITES

ASCITES.—A collection of fluid in the peritoneal cavity recognizable by clinical examination.

Etiology.—The development of ascites may be active or passive. The fluid may accumulate by transudation through the thin walls of abdominal vessels damaged by changes in the chemical or physical properties of their contents; or by exudation from the peritoneal membrane itself as a response to irritation or a result of disease.

Transudation occurs in cases of "venous stagnation" in the portal vein. The obstruction may be due to narrowing of the portal vein or its tributaries by thrombosis or the pressure of enlarged glands, etc.; it may arise in the substance of the liver by compression or obliteration of venous capillaries, as in cirrhosis of the liver; or it may be the result of similar processes acting upon the hepatic veins or inferior vena cava. Finally, the venous stagnation may be due to heart failure. In all these cases an important factor is some degenerative change in the small vessels consequent upon the continued presence of harmful elements. The presence of analogous substances of an irritant nature, combined with general fluid retention, will explain the ascites of kidney disease.

In *exudation* the secretory activity of the peritoneum may be stimulated by mechanical or chemical irritation. The former occurs only in the rare cases of "acute ascites" following an abdominal injury. More often the mechanical irritation is accompanied or overshadowed by chemical irritation. If this is severe, as in perforation of a hollow viscus, acute general peritonitis develops; but when the irritation is less intense and more persistent true ascites may be produced. It is seen especially in cases presenting chronic local or general thickening of the peritoneum: these fall into the clinical groups of perihepatitis, perisplenitis, and polyserositis, in which the peritoneum shares with the pleura, pericardium, and mediastinal tissues in a response to a chronic irritative process of obscure nature. Among infections, in the case of tubercle alone may a chronic non-encysted effusion be produced.

Pathology.—Ascitic fluid varies greatly in composition, but in any individual case the characters of the fluid remain remarkably constant. It is usually pale yellow, clear, or slightly turbid, it does not coagulate spontaneously on cooling, its specific gravity may

vary between 1010 and 1045, but is usually 1020–1035. It contains a varying proportion of albumin and globulin—most in "cardiac" and least in "renal" ascites. Salts similar to those of the blood are present, and cells derived from the blood or peritoneum are to be found, consisting mainly of polymorphonuclear and mononuclear corpuscles and epithelial cells. Occasionally "malignant" cells from tumours invading the peritoneum may be seen. Fat may be present in sufficient quantity to render the fluid milky. Such an appearance is met with in three conditions, viz. chylous, chyloform, and pseudo-chylous ascites.

Chylous ascites is produced by the contamination of ascitic fluid by leakage of chyle from the lacteals as a result of their obstruction or rupture. The obstruction may be due to filariasis, or the pressure of enlarged glands, etc., upon the receptaculum chyli or thoracic duct. Rupture may be traumatic or the consequence of ulceration. The fatty globules are extremely small and are present in the form of an emulsion.

In *chyloform ascites* the fat globules are larger and are derived from the disintegration of abdominal fat or the fatty degeneration of cells in the fluid. Such cells containing fat globules can be found on microscopical examination.

In *pseudo-chylous ascites* the cloudy appearance is due to some body of a proteid nature derived from the degeneration of tissue-cells set free in the fluid. According to Mackenzie Wallis the body is euglobulin—globulin with which is associated a small quantity of lipoids.

Diagnosis.—This may be discussed under three headings:

1. The signs of fluid.
2. The conditions giving rise to similar signs.
3. The differential diagnosis of the cause of the ascites.

1. **Signs of fluid.**—When the amount is small its detection may be impossible. Definite physical signs are given by moderate or large effusions only.

Inspection.—Bulging of the abdomen with prominence of the umbilicus is usual. When the patient is lying down the bulging is evident in the flanks, and measurement shows that the greatest girth is at the level of the umbilicus. If this measurement be taken when the patient is upright, the greatest girth may be below the level of the umbilicus if he has had a pendulous abdomen before the appearance of

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ascites. It is important to notice that the abdominal distension is uniform without any local irregularity of outline.

Palpation provides a very important sign in the "fluid thrill." To elicit this the patient should be lying down; gentle flicking of one flank with the finger-nail sets up a wave of vibration which is transmitted by the fluid across the abdomen, and can be distinctly felt by the palm of the hand applied to the opposite flank. One caution is necessary: the impulse may be conducted by the abdominal wall itself, especially in fat patients. To eliminate this source of error the edge of an assistant's hand should be applied along the linea alba to act as a damper upon such vibrations.

Percussion.—Where fluid is present the percussion note is dull; with the patient lying down, dullness is to be found in the flanks and hypogastrium. Usually the central region of the abdomen is occupied by gut and yields a resonant note. Occasionally the mesentery is shortened and the gut bound down to the posterior wall of the abdomen; if in these cases much ascites be present the resonance of gut usual in the central abdomen may be masked by superjacent fluid.

Shifting dullness is a pathognomonic sign of fluid. It is elicited by percussion of one flank while in the dorsal position and again while in the opposite lateral position. The boundary between dull and resonant areas should be exactly marked out in each case, and, provided that before percussing in the new position a sufficient interval of time is allowed to elapse for the displacement of fluid to occur, a change from dullness to resonance in the flank may be regarded as clear evidence of the presence of free fluid.

Exploration with a hollow needle or trocar and cannula over a dull area will yield conclusive evidence of the presence or absence of fluid in doubtful cases. The method of procedure is described under treatment.

2. Conditions giving rise to similar signs.—

(1) *Fat* in the abdominal wall or its contents may yield a dull note on percussion and a pseudo-thrill as mentioned above. Fat does not, however, give the sign of "shifting dullness." When a small amount of ascites is present in a fat patient its recognition is often very difficult.

(2) "*Colloid cancer*" of the peritoneum is a rare condition in which, usually secondary to or by extension from malignant disease of one of the abdominal or pelvic organs, the perito-

neum is partly converted into, partly filled by, a semi-gelatinous mass of malignant tissue which gives rise to all the signs of fluid. Its recognition is impossible without laparotomy, and its existence is usually unsuspected.

(3) *Encysted collections of fluid* cause local bulging, dullness on percussion, and a fluid thrill. They are to be recognized by observing (a) that the position of the dullness is not typical of ascites, (b) that although the dullness may shift with change of position of the patient its excursion is limited, (c) that some local swelling with asymmetry of the abdomen is present.

The collections of fluid which must be excluded are (a) a distended bladder, (b) a pregnant uterus with or without hydramnion (c) ovarian cysts, (d) other intra-abdominal cysts, e.g. pancreatic and mesenteric, (e) localized peritoneal effusions enclosed by peritoneal adhesions. The history of the case and some departure from the typical signs are usually sufficient to exclude ascites. It must not be forgotten that the coexistence of localized cysts, e.g. ovarian, and ascites is not uncommon.

3. **Recognition of the cause of the ascites** may be rendered simple by the history of the patient, e.g. in cases of gross heart failure or renal disease, especially of the parenchymatous variety. When ascites has developed insidiously and without definite signs of general or local disease recognition is more difficult, and may be impossible, but the most reliable indications are to be derived from the characters of the fluid.

A passive origin is suggested by low specific gravity, absence of spontaneous coagulation, low albumin content, and scarcity of cellular elements.

An active origin, indicating chronic peritonitis, is suggested by high specific gravity, the occurrence of spontaneous coagulation, high albumin content, and the presence of many cells. The discovery of typical malignant tissue-cells or excess of small lymphocytes has an important significance as pointing to a malignant or tuberculous origin respectively. Rapid and repeated recurrence of effusion after tapping, in the absence of clear evidence of venous engorgement, points to a peritonitic origin.

Treatment.—Apart from that of the causal condition, this resolves itself into measures calculated to inhibit the formation and to promote the absorption or elimination of the effusion. The former of these objects may be

helped by limitation of the intake of fluid, and, in renal cases especially, by the exclusion of salt from the dietary. The latter object may be attained by the employment of eliminative drugs or by operative measures. The drugs which have a special reputation in this connexion are the saline purgatives, mercury by the mouth or by local inunction, and potassium iodide. Where there is a possibility of a syphilitic infection the latter drugs should be employed actively; in ascites associated with syphilitic liver disease their action is most valuable, and there is evidence to show that they are useful also in ascites due to chronic peritonitis of obscure origin. Drastic purgation, except as an occasional measure, is inadvisable: the use of aloes is contraindicated owing to its congestive effect upon the pelvic viscera. The value of diuretics is not great, though benefit may sometimes follow the use of theocine sodium acetate in doses of 5-10 gr. thrice daily.

Operative treatment includes simple aspiration and the more active measures of laparotomy, hepatopexy, omentopexy, and lymphatic drainage. Aspiration is best carried out by means of a Southey's tube inserted in the mid-line of the abdomen or in the flank, and connected by a fine rubber tube filled with saline or antiseptic solution to a vessel under the bed. The trocar should be inserted in an area which is dull on percussion. The bladder should be known to be empty before a trocar is used. In the mid-line there is no danger of wounding a vessel, in the flank the line of the deep epigastric artery (from the mid-point of Poupart's ligament to the umbilicus) must be avoided. The danger of wounding the cæcum is slight. Provided the rubber tubing is filled with solution the ascitic fluid will drain away by siphonage. There is a danger of collapse if the fluid is removed too quickly; to guard against this the patient should be kept in the recumbent position, and a firm abdominal binder should be worn during the process of drainage and tightened from time to time as the fluid drains away.

Occasionally simple aspiration may produce a cure; much more often its early and frequent repetition is necessary.

Simple laparotomy with rapid removal of the fluid has been followed by cure; this is more likely to occur if measures are adopted to promote the formation of adhesions between the liver or omentum and the anterior abdominal wall. This object may be attained by

simple "scrubbing" of the approximate areas or by grafting of omentum between the layers of the abdominal wall. The patients who are most suitable for such treatment are those who have already survived four or five simple tappings and who show no evidence of disease of heart, lungs, or kidneys. Such patients form a small minority of those who present ascites. Lymphatic drainage, by the insertion of silk threads from the peritoneal cavity into the subcutaneous tissues of the thigh, or by the formation of a permanent foramen in the femoral canal, has recently been attempted, but its attainment has been only partially successful.

C. E. SUNDELL.

ASIATIC CHOLERA (*see* CHOLERA).

ASPHYXIA. — The term asphyxia (which etymologically means pulselessness) is applied to the condition in which the supply of oxygen to the blood is reduced. Symptoms of asphyxia will be produced by any cause which prevents the proper oxygenation of the blood, and death will, of course, result when the supply of oxygen falls below the minimum necessary for the maintenance of life.

The **symptoms** of asphyxia may be divided into three groups:

In the *first stage* there is increased respiratory activity, and the muscles of extraordinary respiration are called into action; the expression is intensely anxious; the lips become blue, and the eyes are prominent.

The *second stage* is an exaggeration of the first, from which it is not separated by any definite line of demarcation. The violent respiratory efforts become convulsive, and general convulsions of the body supervene.

In the *third stage* the respirations are slow, irregular, and gasping, the muscles become flaccid, the conjunctivæ are insensitive and the pupils widely dilated, there is insensibility, breathing ceases, and death ensues. The heart may beat for some seconds after respiration has stopped, and resuscitation may be effected by appropriate treatment.

The chief forms of violent death in which asphyxia is the proximate cause are drowning, suffocation, hanging, strangulation, poisoning by CO, CO₂, and some other gaseous poisons. But the condition is also frequently met with in disease, and may occur in croup, œdema glottidis, lung diseases, affections of the respiratory centre and nerves, embolism and thrombosis of the pulmonary vessels, etc.

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Post-mortem appearances.—In the violent forms of asphyxial death the symptoms are by no means constant, and sometimes not even typical.

External.—There is pronounced lividity. The lips may be pale blue to almost black, the face distorted and livid, the conjunctivæ congested, the tongue partially protruded, and there may be bloodstained froth at the mouth and nostrils; but frequently the face is calm and placid, and the colour normal except for some blueness of the lips.

Internal.—The blood is dark, and remains fluid for an unusually long period. The venous system in general is gorged with dark blood, but great variations are met with in individual cases in the condition of the brain and meninges, the lungs, and the right side of the heart. The left side of the heart is empty. The engorged lungs yield on section a copious bloody, frothy fluid. Punctiform hæmorrhage (*Tardieu's spots*) may be found on the pleura, and some of the air-cells may be ruptured (emphysema). The mucous membrane of the trachea is often cinnabar-red in colour.

DROWNING

A person may be drowned, of course, without the whole body being immersed, nothing more being necessary than that air should be prevented from entering the lungs by the mouth and nose being covered with the fluid. Epileptics and intoxicated persons have been drowned by falling face downwards into small pools of water, and an insane person has been known to drown himself by submerging his head in a sink filled with water.

Time required for death in drowning.—Experiments on dogs show that they are killed by one and a half minutes' complete submersion, whereas they recover after simple deprivation of air for four minutes. That drowning is more rapidly fatal than simple deprivation of air is due to fluid being aspirated into the minute air-tubes and cells of the lungs. A human being is generally asphyxiated by less than two minutes' complete submersion, but there is on record the case of a woman who by practice had attained the power of remaining submerged for two and a half and even three minutes. The smaller the quantity of fluid which has penetrated to the lungs, and the smaller the amount of froth in the air-tubes, the greater the chance of resuscitation, and those who enter the water with their breathing practically in abeyance

(e.g. in a state of insensibility) will take longer to die than those who make violent respiratory efforts and thus aspirate water into the air-passages and lungs. The amount of submersion, whether complete or partial, must also be taken into account.

Treatment of persons apparently drowned.—Cleanse the mouth and nostrils of all mud or other foreign material; strip the body to the waist, and dry it by rubbing with warm towels; proceed with artificial respiration (*see* RESPIRATION, ARTIFICIAL); apply warmth by hot flannels or blankets, hot-water bottles, etc., and friction. Attempt to induce reflex efforts at inspiration by the application of the vapour of ammonia, aromatic vinegar, etc., to the nostrils. When breathing has been restored, put the patient to bed and apply warmth to the surface of the body. Small quantities of stimulant (warm water with a little brandy) may be given when the power of swallowing has returned.

Cause of death and post-mortem appearances.—The proximate cause of death in drowning is asphyxia. Both external and internal post-mortem appearances vary with the length of time the body has been in the water and with the interval between its removal from the water and the examination. The examination should be carried out as soon as possible after the body is recovered, as putrefactive changes come on rapidly and interfere with the characteristic appearances.

External appearances.—The surface is cold and pallid, and the condition of cutis anserina or goose-skin may be present, also retraction of the penis. The contracted state of the skin is evidence that molecular life was present when the body entered the water and is due to the action of cold. The skin of the palms ("washerwoman's hands") and soles is sodden, bleached, and wrinkled, but this is the result merely of remaining in the water, and has nothing to do with the cause of death. The body and limbs are usually relaxed, but post-mortem rigidity comes on rapidly, and a convulsed attitude due to instantaneous rigor may be met with. Instantaneous rigor will also account for objects—such as weeds, twigs, gravel, etc.—sometimes found grasped in the hands. Note should be made of any wounds or other marks of violence, but it should be remembered that abrasions on the hands and fingers may be the result of grasping at any object within reach. Usually, the face is pale and calm, the eyes are closed or partially

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opened with the pupils dilated, the tongue is usually dusky in colour and perhaps protruded beyond the line of the teeth, and a fine froth is present at the mouth and nostrils. A rosy hue of the lips and of the skin over the malar prominences is sometimes observable when drowning has taken place in frosty weather. Livid discolorations may be found to a greater or less extent over the body. The two external appearances which strongly indicate death by drowning are the presence (1) of a fine foam at the mouth and nose, and (2) of weeds, etc., firmly grasped in the hands.

The *internal appearances* found are those of asphyxia, but some additional characters assist in determining that death was due to drowning. The lungs are seen to be fully distended ("balloon lungs") and bulge forwards on opening the thorax; their elasticity is lost and they retain impressions made on them by the fingers; on section there is an escape of frothy, bloody liquid, and on squeezing the organ a fine, watery, bloodstained froth can be expelled from the air-sacs and smaller air-tubes. A similar froth is found in the bronchi and trachea. This froth is due to violent respiratory efforts after water has been aspirated into the air-passages. These appearances are found most pronounced when the body is examined soon after death. The stomach commonly contains fluid corresponding with the medium in which the body was drowned.

Was drowning the cause of death?—Not all bodies recovered from the water are drowned bodies. A person may die from apoplexy and his body fall into the water, or a body may be thrown into the water to cloak the commission of a homicide. The signs which should be relied upon, though they need not all be present in a particular case, are:

1. The presence of substances (associated with the *locus* of the drowning) grasped in the hands or lodged under the nails.

2. The presence of a fine foam at the mouth and nostrils.

3. Ballooning of the lungs and the escape of a watery, bloodstained froth on section. If the person was unconscious on falling into the water and the respiratory efforts were feeble, these appearances will not be well marked.

4. The presence in the stomach of liquid of the same nature as that in which the body was immersed. Floating material, such as weeds, etc., may also be present.

Accident, suicide, or homicide?—*Accident* is

by far the most frequent cause of drowning. In the case of *suicide*, attendant circumstances—such as weights tied round the neck, heavy objects in the pockets, mental state, and motive for the act—will serve as indications. The question of *homicide* will arise in such circumstances as the following: (1) The presence of evidence of a struggle near the water from which the body has been recovered. (2) The finding in the hands of the drowned person of hair or portions of clothing which do not correspond to his own. (3) The presence of lesions due to violence while the signs of drowning are absent.

Each case must be decided after considering all the circumstances. It has already been pointed out that the characteristic lung appearances will not be very distinct when the breathing has been feeble and there has been no violent respiratory effort. Marks of violence may have been produced post mortem; in navigable waters, bodies, when they float, may sustain extensive injuries from the propellers of passing vessels, or by other means. The question of the ante- or post-mortem production of these injuries and of their ability to cause death will then have to be decided (*see INJURIES FROM THE MEDICO-LEGAL STAND-POINT*).

SUFFOCATION

Suffocation is that condition in which air is prevented from entering the lungs, not by constriction of the windpipe but by some external cause which blocks the mouth and nostrils or prevents the movements of the chest, or some internal cause which obstructs the air-passages. It may result from natural disease as well as from accident, suicide, or homicide.

Death by suffocation may be caused by simple privation of air from want of proper ventilation; "smothering," which consists in the mere covering of the mouth and nostrils in such a way as to prevent breathing; the breathing of poisonous gases such as CO or CO₂; fixation of the chest as the result either of disease or of external pressure, as when a person is pinned down by falling debris, or wedged in a panic-stricken crowd; obstruction of air-passages by disease, e.g. tumours, an inflammatory swelling, the bursting of an abscess or an aneurysm, the impaction of foreign material in pharynx or larynx.

Treatment of suffocation.—If the cause is removed, respiration may become re-established spontaneously; but if there has been

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complete deprivation of air for five minutes, death ensues. When a person has been choked by a mass of food or other solid material, the indications may be for immediate tracheotomy instead of losing time in attempts to remove the obstruction. After the cause of the asphyxia has been removed, artificial respiration should be instituted.

Post-mortem appearances.—As the proximate cause of death is asphyxia, the signs of that condition will be expected.

External.—Livid patches over the skin, lividity of the lips, congestion of the eyes, and mucous froth about the lips and mouth may be met with, but in many cases the facial appearance is normal except for a bluish tint of the lips and the mucous membrane of the mouth. In children, lividity of the finger- and toe-nails is common.

Internal.—The ordinary signs of asphyxia will be most evident in cases in which death has been produced slowly. The appearances, however, are not constant, and in many cases there will be little or nothing to suggest the cause of death. Special attention should be paid to the mouth and air-passages to discover if foreign bodies are present.

In the *overlying of infants* there may be flattening of the nose, or one cheek may be pale and flattened from pressure, or the weight of the mother's arm on the child's chest may have been the cause of the asphyxia. Apart from external signs of pressure, all the signs of asphyxia here found are consistent with death from convulsions. These cases occur usually in infants who sleep with their mother, father, or other adult. The great majority of deaths are the result of accident and not of criminal design. A woman falls asleep while suckling her child in bed, and the infant is smothered by pressure against her breast; or suffocation may result from the child's head slipping beneath the bedclothes. When signs of pressure are absent it is only possible to say that death was due to asphyxia, and not how it was produced.

The question of criminal responsibility will have to be considered in cases in which the person who was in bed with the child was under the influence of alcohol; in this connexion Sect. 13 of the Children Act, 1908, is worth quoting in full:—"Where it is proved that the death of an infant under 3 years of age was caused by suffocation (not being suffocation caused by disease or the presence of any foreign body in the throat or air-passages

of the infant) whilst the infant was in bed with some other person over 16 years of age, and that that other person was at the time of going to bed under the influence of drink, that other person shall be deemed to have neglected the infant in a manner likely to cause injury to its health within the meaning of this part of this Act."

Accident, suicide, or homicide?—The chief points requiring attention are the age and the condition of the deceased, the presence of marks of violence, the nature of the substance causing death, and the position of the body.

Homicidal cases occur chiefly in children, the aged and infirm. Accidental suffocation may obtain when, e.g., a person is choked by food, when an intoxicated person so falls that access of air to his mouth and nostrils is prevented. Marks of violence will indicate a struggle, and, unless the person was insensible from disease or intoxication, resistance would be expected in homicidal cases. The obstruction of the air-passages of an infant would point clearly to homicide, while the presence of false teeth or food in the air-passages of an adult would indicate accident. The position in which the body is found may also allow of deductions being made.

HANGING, STRANGULATION, AND THROTTLING

Hanging is that form of death in which the body is wholly or partially suspended by the neck, so that death is caused by the weight of the body acting upon the constricting ligature. In *strangulation* the constricting force is applied circularly round the neck and by some other means than the weight of the victim's body. In *throttling* (manual strangulation) the constricting force is the hand or fingers applied to the windpipe.

Hanging.—The time required to produce insensibility and death depends on the situation of the ligature and the severity of the constriction (i.e. whether the body is or is not completely suspended). Fleischmann, who experimented on himself, found that when the cord was placed between the chin and the hyoid bone, and moderately tightened without perceptibly interrupting respiration, suspension for two minutes was possible. When the pressure was applied on the windpipe the effect was instantaneous; and when applied between the hyoid bone and the thyroid cartilage, or on the hyoid bone itself, the period during which breathing was possible was very short.

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The common cause of death in hanging is occlusion of the air-passages, but other factors may play a part. Stasis of the cerebral circulation from pressure on the main venous trunks in the neck is one of the results of suspension; and a man on whom tracheotomy had been performed is known to have committed suicide by hanging, the ligature being placed above the site of the tracheotomy wound. At the post-mortem the vessels at the base of the pons and medulla were found engorged with blood. Injury to the vertebral column and spinal cord is caused by judicial hanging and when a suicide has given himself a drop.

Treatment.—The body, supported to prevent it from falling, should at once be cut down. Artificial respiration is then started, and the vapour of ammonia, etc., may be employed, as in cases of drowning. About a pint of blood may be removed by venesection to relieve the venous stasis, and mustard poultices should be applied over the heart, to the calves of the legs, etc. Hypodermic injections of ether may be given, and electricity may be tried over the course of the phrenic nerves. Bodily warmth should be maintained by hot blankets and hot-water bottles.

Post-mortem appearances, external.—These are very variable, and the only characteristic appearance is the mark produced by the ligature. Commonly the face is pale and placid, the eyes are shut or partially open and the pupils dilated, the lips bluish in colour, and the tongue normal in position or protruded slightly in front of the teeth. Only exceptionally is there found great lividity of the face with protrusion of the eyes and tongue and clenching of the hands. Involuntary discharges of fæces, urine, and seminal fluid are described by some writers, but these may occur in sudden and violent death from any cause.

Special examination of neck.—The mark or marks of the ligature must be carefully examined. The position and direction of the marks will vary with the mode of suspension. If the ligature has been tied tightly round the neck or if a running noose has been used, the mark will tend completely to encircle the neck. Where a fixed loop has been employed, it will occupy the highest position which anatomical relations allow. Generally the mark will be oblique, passing upwards behind the ear, where it is lost. If the noose should be under the chin the mark may be circular, the lower

jaw preventing the ligature from rising upwards as it usually does behind. The deepest part of the mark is on the side opposite to the knot, the position of which is indicated by an irregular area of compression or by the mark being interrupted. If the ligature has been passed twice round the neck a double mark will be found, one being more or less circular and the other oblique. Multiple marks may be present when a ligature composed of a number of strands of cord has been employed.

The width of the mark will depend on the nature of the ligature, and a pattern of the material may be left on the skin. The ligature should always be examined and compared with the marks on the neck.

The colour of the mark varies considerably. In some cases there is simply a pale furrow, in others a bluish- or brownish-red colour is met with, while in yet others the mark is of the colour and consistency of parchment. The parchment-like mark is due to abrasion of the cuticle and desiccation, and only comes on some hours after death. Ecchymosis, while not commonly met with in suicide, is of importance as evidence of vital reaction.

Post-mortem marks, internal.—Ecchymosis, injuries to arteries and muscles, fracture of the larynx, the hyoid bone, and spinal column are rare, and will only be met with when considerable violence has been employed in the hanging. The general internal appearances are those of asphyxia, and the vessels of the brain are usually found congested.

Was death due to hanging?—A body may be hung after death to simulate suicide. When this question arises, strict attention must be given to every aspect of the case, and a post-mortem examination is absolutely essential. It has been shown experimentally that the mark usually found on the neck of a person who has committed suicide by hanging can be produced on a dead body which has been suspended within two hours after death. If the internal appearances point to asphyxia, special attention should be paid to the marks on the neck and to the ligature, as the person may have been strangled and the body thereafter suspended. When marks of violence are found, the possibility of their being sufficient to account for death should be considered.

Accident, suicide, or homicide?—The great majority of deaths from hanging are *suicidal*. No hard-and-fast rules can be laid down, and the question will have to be determined according to the circumstances attending each

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case. In suicide the body may be suspended entirely above the ground, or the major portion of the trunk may be resting on the ground. A double mark, one circular and the other oblique, is not inconsistent with suicide, as the ligature may have been passed twice round the neck.

Homicidal hanging is rare, and, except in the case of a child or of an older person who is insensible, is difficult to accomplish single-handed without leaving marks of violence. But a suicide may inflict wounds on his body or take poison before hanging himself. The hands of the deceased should be inspected for injuries received while defending himself.

Cases of **accidental hanging** have been recorded, as when a boy slipped while climbing a tree, and his jacket, which was buttoned at the neck, turned up round his neck and suspended him.

Strangulation.—The treatment of persons strangled is on the same principle as in cases of hanging.

Post-mortem appearances.—The mark produced by the ligature is generally circular. It may be found on any part of the neck, but is generally round the windpipe below the larynx, in contrast to the position above the larynx found in hanging. In homicidal strangulation the force employed is usually considerably in excess of that necessary to cause death, and evidence of violence will be found on the skin and underlying structures. When a soft ligature has been employed there may be no marked furrow or ecchymosis, merely a slight depression of the skin.

Both the external and internal appearances of asphyxia are generally more distinct than in hanging, and it is more common to find the face swollen and livid, the eyes prominent and congested, the tongue swollen, congested, and protruded, and bloody froth at the mouth and nostrils. Tardieu's spots (*see* p. 125) are sometimes found beneath the skin of the face, neck, and chest.

Accidental cases of strangulation are not uncommon, and suicide by this means occurs occasionally, and can be effected by tying a ligature round the neck and twisting it tight by means of a stick. Homicidal strangulation is much easier of execution than homicidal hanging, and therefore occurs more frequently. The points to be considered in coming to a conclusion in any particular case are similar to those discussed in connexion with hanging.

ASPHYXIA NEONATORUM

Throttling.—In manual strangulation the marks will usually be found on the lateral aspects of the neck, and there are generally a thumb-mark on one side and finger-marks on the opposite side. Comparatively slight force will close the vocal cords or force the base of the tongue against the posterior wall of the pharynx, but a murderer generally uses great force, and fractures of the larynx and hyoid bone may be found, as well as extravasations of blood into the subcutaneous tissue.

A. ALLISON.

ASPHYXIA NEONATORUM.—A condition of the new-born child in which there is no attempt to breathe immediately after expulsion from the birth canal. Clinically, there are two types, named, according to the appearance of the child, *asphyxia livida* and *asphyxia pallida*.

Etiology.—The commoner causes are defective oxygenation of the foetal blood, (a) by pressure on the funis, as in labour with prolapsed cord or after-coming head, (b) by premature detachment of the placenta, as in placenta prævia, (c) by pressure on the thorax and neck after delivery of the head. A less frequent cause is pressure upon the head, whether by forceps, by prolonged passage through a contracted pelvis, or by intracranial hæmorrhage. Moderate continuous pressure seems to exert a more malign influence than that which is more severe but intermittent. It probably acts by paralysis of the vagal respiratory centres.

Symptomatology.—Progressive slowing of the foetal heart during delivery is strong evidence of approaching asphyxia. Temporary slowness is due to a rise in foetal blood-pressure, but when progressive it is a sign of stimulation of the vagal centres (cardio-inhibitory) by imperfectly oxygenated blood. In less common cases the heart-beat may be very rapid and faint. Hence, a fall below 110, or a rise above 160, during delivery, between the pains, is strong evidence of foetal distress. Other signs are spasmodic efforts at inspiration made during a breech delivery while the head is yet in the pelvic canal, and the passage of meconium in cases other than breech presentations. It is most important to recognize impending foetal asphyxia, before delivery, by a close watch on the foetal heart, so that measures may be taken to deliver the child alive. After delivery the two varieties mentioned above, blue and white asphyxia, are recognizable.

Blue asphyxia, the milder type, is characterized by the cyanotic hue of the body and the presence of some muscular tone. The face is blue and congested, and the mouth exudes mucus. The rest of the body is usually less cyanosed but dark red, and the limbs are not completely flaccid, while some skin and mucous-membrane reflexes remain. The heart-beat is distinct but slow, and can be felt in the cord near the umbilicus. It is common for the child to make spasmodic gasps for breath, which are usually unsuccessful on account of the presence of mucus in the trachea and bronchi. If, however, air is inspired by such efforts, they become more frequent and less spasmodic, until a cry is uttered and normal breathing becomes established.

White asphyxia, the severer type, is characterized by the white, bloodless appearance of the whole body, which is entirely toneless and limp. The child appears to be dead, except that careful examination reveals the presence of a feeble heart-beat, either very slow or irregular. The cord is pulseless. There is no effort to breathe, and gasps are not made until after restorative measures have been applied. The reflexes are absent. The only sign of dormant life is the feeble heart-beat.

Treatment. Blue asphyxia.—Treatment is to be directed towards clearing the air-passages of mucus, and stimulating the respiratory centre. Hold the child up by the ankles, to allow mucus to drain away, and slap or rub the spine. Dash drops of cold water over the trunk. Should the pulse be felt strongly in the cord, no anxiety need be felt; but if the child does not breathe, ligature and cut the cord, and apply Sylvester's method of artificial respiration (see RESPIRATION, ARTIFICIAL), taking care to swab the mouth clear of mucus. Spontaneous breathing is rapidly established.

White asphyxia.—The most prominent symptom to be dealt with is cardiac weakness, and treatment should be directed towards restoring the heart's action by artificial respiration and the administration of stimulants. The general condition is one of shock. First, attempt to remove mucus and liquor amnii from the air-passages by swabbing; then, lifting the child gently, convey it immediately to a hot bath, 105° F. Next, carry out artificial respiration in the bath by Sylvester's method, for which an assistant is almost necessary. Artificial respiration should be undertaken slowly and steadily, without adopting repeated changes

of method. Meanwhile, the temperature of the bath should be maintained by adding hot water. Finally, during the course of artificial respiration an assistant may rub the chest and gums with a few drops of brandy, or a hypodermic injection of strychnine ($\frac{1}{200}$ gr.) or pituitary extract (2 min.) may be given beneath the clavicle. If the throat is full of mucus, this should be removed by suction through a small metal catheter. Direct insufflation of the lungs has little to recommend it, as it seldom succeeds where artificial respiration fails, and there is grave danger of injuring the glottis or rupturing pulmonary vesicles. Treatment should be continued for as long as the heart beats, for there is always some hope, but most cases will respond within forty-five minutes, if the heart is fairly regular or if there are any spontaneous gasps. After such resuscitation, great care should be taken during the first week, for the child is often feeble and may die from imperfect expansion of the lungs (*atelectasis*). A. W. BOURNE.

ASPIRATION (see ASCITES; PLEURISY).

ASPIRATION PNEUMONIA (see Broncho-pneumonia, under PNEUMONIA).

ASTASIA-ABASIA (see HYSTERIA).

ASTEREOGNOSIS.—This term is applied to loss of the power of recognizing the shape of objects by touch, or by the movement of the hand or any part of the body over them, and occasionally in a wider sense to inability to distinguish other physical properties in them, as consistence, roughness, and smoothness. Stereognosis is not an elementary sense or simple sensation, but is the result of judgments based on the association of various cutaneous and proprioceptive perceptions, as the sense of position and of passive movement, the localization of cutaneous stimuli, and the discrimination of two or more simultaneous contacts. It is never disturbed when all these qualities of sensation are normal, but is affected by the loss of any one of them. Astereognosis consequently results when disease, especially in the cerebral cortex, the median fillet, or the dorsal columns of the cord, involves the centres or paths concerned in these perceptions. It occurs in tabes, the combined degenerations of the cord, occasionally in disseminated sclerosis, and on the paralysed side in unilateral spinal lesions.

GORDON HOLMES.

ASTHMA

ASTHENIO BULBAR PALSY (see MY-ASTHENIA GRAVIS).

ASTHENOPIA (see REFRACTION AND ACCOMMODATION, ERRORS OF).

ASTHMA.—A disorder of respiration, leading to recurrent spasmodic attacks of dyspnoea from reflex causes.

Etiology.—As appears from the definition, asthma is not a disease but an objective symptom. The disorder of respiration comes about somewhat as follows: In normal breathing the respiratory movements depend upon the respiratory centre in the medulla, and the phrenic and other nerves supplying the inspiratory and expiratory muscles. Afferent impulses to the centre are conveyed in normal respiration not by afferent nerves, but by carbonic acid in the blood-stream. It proceeds, therefore, independently of afferent nerves. Afferent impulses, however, can reach the respiratory centre, and such impulses may explain the asthmatic attack. They may come from many parts of the respiratory tract, from the nasal mucous membrane or from the bronchi, and also from the stomach and intestines, and doubtless many other surfaces. Psychical influences, too, are of definite importance.

If the vagus is stimulated, contraction of the circular muscles of the bronchioles occurs, and a reflex stimulus to the vagus can be provoked by irritation of the nasal mucous membrane. The state induced by this method is said to be indistinguishable from clinical asthma. It would seem, therefore, that *asthma consists in spasmodic contraction of the circular muscles of the bronchi induced through the vagus by irritation of some mucous surface*. One must suppose that a contraction of the bronchioles interferes with aeration and produces the dyspnoea, which is thus seen to be due to incoordination of the respiratory muscles. Dyspnoea induced in the ordinary way leads to increase in the rapidity and depth of the respiratory movements, but the dyspnoea of asthma is modified by the fact that the inspired air can only reach the alveoli through bronchioles which are spasmodically contracted; therefore the rapidity of respiration is not increased, and is sometimes decreased in spite of the respiratory distress. In this connexion the superior strength of the inspiratory over the expiratory muscles produces definite results: the patient is breathing hard, and his inspiratory muscles can overcome the resistance in the bronchioles better than his expiratory. Con-

sequently there is some over-distension of the chest, the alveoli being more easily filled than emptied.

In addition to the bronchiolar spasm there is also a free secretion of mucus of an extremely viscid character from the mucous membrane, which doubtless adds to the obstruction in the tubes. Whether it is due to the vagal stimulus directly, or follows from the contraction of the muscle, is not clear.

All persons do not suffer from asthma as a consequence of irritation of the respiratory or other membranes. The essential factor in the situation, and one towards which definite treatment should be directed, is a congenitally irritable respiratory centre. Given this irritability, definite lesions of the respiratory and other mucous membranes often coexist from which afferent impulses pass and precipitate the reflex. Such are nasal deformities, nasal catarrh, adenoids, bronchitis, gastritis, and doubtless similar lesions of the abdominal and pelvic viscera. There is reason to think that the turgescence and hypersecretion of mucus in the mucous membrane is of a character allied to urticaria.

Pathology.—Post-mortem records on persons who have died during a paroxysm are few. Berkart has collected seven, and has found the points in common to be dilatation of small and medium-sized bronchi and their obstruction by catarrhal products, infiltration of the peribronchial tissue, congestion of the mucous membrane, emphysema of the alveoli, and dilatation of the heart. The epithelium of the bronchi exhibited various changes, and was in some cases active and in others degenerated and stripped.

Symptomatology.—The chief symptom is spasmodic dyspnoea. This occurs chiefly at night, but may do so at any time. It is sometimes preceded by prodromata of different kinds, comparable to the aura of epilepsy, by which the patients can recognize its imminence. The attack generally begins with a sensation often described as oppression, and this is followed by an increasing difficulty in breathing, aggravated often by alarm and violent efforts on the patient's part. The dyspnoea is often spoken of as expiratory, the impediment to expiration being greater than that of inspiration. The patient cannot maintain the recumbent position, but has to be propped up in bed, sometimes to get out of bed altogether, and in the severe attacks he assumes such positions as enable him to fix his

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shoulders and use all the accessory muscles of respiration to the greatest advantage. In spite of all these efforts the respirations are not increased in rate but rather reduced, chiefly owing to prolongation of expiration. The attack lasts for an indefinite period; two hours is a common length, but it may be much longer. It usually terminates with expectoration of mucus containing small greyish pellets, the "perles" of Laennec. These, if teased in water, can be unravelled into twisted threads known as Curschmann's spirals (see Plate 3); the leucocytes embedded in the expectoration include many eosinophils. With the rejection of this material the spasm is generally relieved.

Physical signs of the chest during an attack.

—The chest is distended and the accessory muscles are seen to be at work. The scalenes and other neck muscles can be felt in contraction. There is some hyper-resonance, and on auscultation and prolonged expiration a considerable variety of intrapulmonary sounds, with a general preponderance of high-pitched sibili, are heard. Long-standing asthma is always associated with bronchitis and consequent emphysema, and often with degenerative changes of the heart.

Cardiac symptoms.—The prolonged respiratory disturbance leads to obstruction in the pulmonary circulation and consequent cyanosis; the pulse becomes feeble and rapid.

Alimentary system.—There is no appetite for food during an attack, but considerable thirst. Salivation is sometimes profuse; there may be nausea and sometimes vomiting. Berkart states that a good deal of air is swallowed during the disturbed respiration, which may be brought up with some relief. During the attacks there is constipation.

Nervous system.—Subjective symptoms are the anxiety and respiratory distress. Headache is common, and cramp and tenderness occur in the thoracic muscles from their prolonged use.

Excretory system.—There is considerable loss of water from all sources. Salivation has been mentioned; there are also profuse sweating and, frequently, considerable diuresis.

Diagnosis is not difficult. Dyspnoea of various kinds arises from heart failure, gross pulmonary lesions, laryngeal or tracheal obstruction, and as a purely functional affection. In all but the last the affection is simply one of defective aeration of the tissues, with accumulation of carbonic acid in the blood and compensatory activity of the respiratory centre.

There is increased rapidity of the respiratory movements, and the accessory muscles are at work, but the effort is chiefly an inspiratory one, expiration being comparatively easy. In hysterical dyspnoea the respirations are rapid and there is no disturbance of the normal ratio of inspiration and expiration. In asthma the essential factor is the obstruction in the smaller bronchi, although it may extend farther up into larger tubes. This presents comparatively little difficulty to the powerful inspiratory muscles, so that inspiration is not much obstructed and is short; the obstruction is chiefly felt in expiration, the inco-ordinate excessive contraction of the bronchioles presenting a difficulty to expiration which is not experienced during inspiration. It is an expiratory dyspnoea, and in spite of the respiratory distress the rate of breathing is decreased instead of increased.

Prognosis. 1. **As to recurrence of the symptom.**—In the majority of cases the prognosis is bad. Asthma frequently starts in childhood and continues at intervals through life. There are patients who apparently "outgrow" it, but they are few. Since the condition depends on an irritable nervous centre, the susceptibility of which varies with circumstances affecting the general health and with psychological influences and degrees of physical exhaustion, the incidence of the dyspnoea is very variable. Again, the reflex source of irritation, whether it be in the nose, bronchi, or stomach, varies with the many different conditions which affect the respiratory tract, such as temperature, humidity, dust, and the prevalence of the organisms causing catarrh, so that asthmatics may experience long periods of immunity or continuous periods of suffering, according to accidental circumstances. Inasmuch as irritable nerve-centres and respiratory affections are difficult to treat, complete or continuous immunity is seldom attained; but where the indications for treatment are clear and systematically attended to, much may be done—the frequency and severity of attacks will at any rate be diminished.

2. **As to life.**—Death in the asthmatic spasm is rare, but, as stated under pathology, it is frequent enough for records of seven cases to exist, though post-mortems are rarely obtained in private practice, so that the risk of a fatal termination is not wholly negligible.

The existence of asthma doubtless shortens life from general wear and tear, interference with normal sleep, and distress. It is closely

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associated with bronchitis, a vicious circle existing between the two, asthmatics of long standing nearly always suffering from chronic bronchitis; thus, the limitations of activity and powers of resistance of asthmatics are the same as those of chronic bronchitics. They are likewise exposed to the cardiac strain arising from chronic lung disease, and hence frequently suffer from shortness of breath of myocardial origin in addition to the reflex dyspnoea of asthma. It may be said, therefore, that though asthma itself rarely causes death, it shortens life.

Treatment.—The treatment of asthma demands attention to several points, and that of the paroxysm, though urgent, is of little consequence in comparison with its prevention. Every rational method of treatment requires knowledge of the causes of the conditions which it is required to avoid, and where actual knowledge is unavailable it is necessary to find a working hypothesis. The hypothesis held here as to the cause of the symptoms admits of a definite line of treatment, which leads to beneficial results when the underlying morbid conditions can be identified and treated.

The first indication is to search the respiratory tract for *sources of reflex irritation*. In the nose, bony obstruction, rhinitis, and adenoids should be sought for; in the throat, enlarged tonsils and pharyngitis; and in the lungs, infective conditions, more especially chronic bronchitis. Morbid conditions in the nose require surgical treatment. It is, of course, impossible to guarantee that the removal of enlarged turbinal bones or of polypi will cure the asthma, but when these defects exist they obviously cannot be neglected in any case of interference with the respiratory function. There are failures, no doubt, but there are very many cases of asthma which are greatly benefited by surgical treatment of nasal abnormalities.

With regard to the treatment of the *bronchial condition* there are several points to be borne in mind. The secretions in asthma are of a very viscid character, at any rate in the affected bronchi, although watery secretions are common in the mouth during an attack, witness the profuse salivation which commonly occurs. An attempt should be made to deal with this viscosity. Assuming that it can be diminished by lengthening the coagulation time of the blood, which is associated with a decrease in its viscosity, an attempt should be made to remove some of the calcium from activity in

the blood, since upon this substance coagulation depends. And just as it is possible by washing out a sinus with citrate of soda to increase the fluidity of the secretion, so by the use of citric acid internally can something be done to loosen viscid bronchial secretions. This action is increased by the use of potassium iodide. A useful prescription is the following:

Ry Pot. iod. gr. iii.
Acid. cit. gr. xx.
Syrupi ʒii.
Aq. chlorof. ad ʒi.
T.d.s.

With regard to *specific* treatment of the bronchitis, much can be attained by means of vaccines. It seems that some organisms rather than others have the power of inducing asthma. In my own experience, infections with organisms of the coli group are more frequently associated with asthma than infections with cocci (pneumococcus, streptococci, micrococcus catarrhalis, etc.). These vaccines are quite useless to abort an attack, but in a large number of instances their prolonged use leads to definite diminution in frequency and severity. Vaccines should be made from the organisms from the patient's own sputum, selecting any coliform bacilli which may be present, and also predominant organisms of other types. It is best to begin with small doses of 10 millions, doubling these at weekly intervals until a dose of about 50 millions is reached. This is, of course, only a generalization, and patients may be found who can take much larger doses with benefit and others who can only take much smaller ones. After about a month of weekly injections the intervals may be prolonged to a fortnight, then to three weeks, four weeks, and so on.

Some patients find their oppression begins to recur after about a month and then require further treatment. There are occasional cases in which treatment is persisted in for many months with apparent cure; at any rate, the patients have remained quite free from attacks for periods of two or three years. In nearly all cases the treatment must be protracted, and it is hardly worth while to embark upon it unless the patients are prepared to give it at least a three-months' trial. Very definite perseverance on the part of both doctor and patient is essential.

The *climatic* treatment is that of bronchitis. Patients should avoid damp and draughts and the risk of infection from other persons, and, if possible, should winter in warm climates.

counsel of perfection with people who have to earn their living. Asthmatics vary much with regard to the climate that suits them; in my own experience most of them suffer severely at the sea, and I have reason to think that many are extremely susceptible to damp atmospheres generally.

With regard to the *irritable nervous centre*, benefit may be obtained by the use of sedative drugs, of which the most effective are the bromides. In persons who are liable to attacks when worried or overworked, I have found a dose of sodium bromide, potassium bromide, and ammonium bromide, 10 gr. of each, at bedtime, of great value.

When investigating asthma in hospital out-patients, I found that attacks were much more frequent on Saturday and Sunday nights after the unusually heavy meals taken on those days. The indication is to regulate the diet and treat any dyspeptic symptoms according to the individual case.

Treatment of the paroxysm.—The object here is to relieve the spasm of the bronchioles. The most effective method of stopping the paroxysm is the injection of morphia, of which $\frac{1}{4}$ gr. is generally sufficient. It is commonly stated that morphia used for the relief of asthma does not induce the morphia habit, but I know of some grave instances to the contrary, and should be very reluctant to allow the patients to inject themselves. For the same reason cocaine should be absolutely excluded; but a drug which is not liable to abuse and from which many patients get great relief is adrenalin, 5 min. of a 1-in-1,000 solution. Less drastic remedies may be tried first. Some patients are relieved by the inhalation of the fumes of stramonium or other antispasmodic powder. The following is a good example of such a powder:

Ry	Pulv. stramon. fol.	} 55 3ii.
	Pulv. lobel.	
	Pulv. thes. nigr. fol.	
	Pot. nit.	

Half an ounce of the powder should be ignited on a tin plate, and the fumes inhaled. Others obtain more relief from the inhalation of turpentine, ether, or chloroform. When attacks are frequent, the addition of tincture of stramonium (10 min.) to the citric acid and iodide mixture recommended above sometimes gives good results.

D. W. CARMALT-JONES.

ASTIGMATISM (see REFRACTION AND ACCOMMODATION, ERRORS OF).

ATAXIC PARAPLEGIA.—This name was originally applied by Gowers to a condition in which ataxia is combined with weakness and spasticity of the limbs, and particularly of the legs. The disturbance of gait was regarded as its most typical feature. It was characterized by unsteadiness, staggering, and difficulty in maintaining balance, especially in the dark or when the eyes were closed, as well as by rigid ungainly movements of the legs and an inability to raise the feet properly from the floor. The reflexes were generally exaggerated, but sphincter disturbances or other symptoms of spinal disease appeared only late.

Experience has, however, shown that there is no separate clinical entity or pathological condition that corresponds to the disease described by Gowers. The symptoms he assigned to ataxic paraplegia occur in different nervous diseases in which the pathological lesions produce both spastic paresis and ataxia of movement. In combined degeneration of the cord the lesions of the lateral columns cause weakness and rigidity of the limbs, that of the dorsal columns loss of the sense of position and consequently ataxia and Romberg's sign, while degeneration of the spino-cerebellar tracts may be responsible for ataxia independently of concomitant sensory disturbances. When tabes is associated with a mild myelitis, meningo-myelitis, or degeneration of the pyramidal tracts, the gait may be both spastic and ataxic. This condition is also occasionally seen in general paralysis and Friedreich's disease, but the most common cause of a spastic ataxic gait is certainly disseminated sclerosis.

GORDON HOLMES.

ATAXY.—A loss of co-ordination and a resultant awkwardness in the performance of a movement, without co-existing paralysis of the muscles involved.

To understand the mechanism of the disturbances leading to ataxy, it is necessary to recall the muscular co-ordinations involved in the performance of a voluntary movement. The execution of a voluntary movement, no matter how simple its nature, involves the participation of a number of muscles other than those primarily concerned in displacing the limb in the desired direction. Three types of muscular action may be distinguished. In the first place, there is contraction of the muscles directly concerned in the movement of the joint, which we may call the prime movers. The rapidity and force of their contraction are

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adapted to the movement to be effected. The second type is represented by the antagonistic muscles, or those whose active contraction would, if unopposed, cause movement of the joint in the opposite direction. If these are in a state of active contraction when the movement is initiated their contraction is inhibited. If they are in the normal state of tonic contraction, one of two things may happen: when the movement is carried on against resistance, as in lifting a weight, their tonus is inhibited, as its persistence can only impede the effective execution of the movement; on the other hand, when the resistance is negligible their tonus is increased in order to exercise a steady influence on the movement and prevent over-action. Thirdly, in order to limit the movement and to prevent displacement of the contiguous parts of the body, fixation of the neighbouring joints is brought about partly by active contraction of the synergic muscles and partly by an adequate increase of tonus in the postural muscles that may be concerned. It is obvious, therefore, that the performance of even the simplest voluntary movement depends upon the active contraction, inhibition, or increase of tonus of a large number of muscles, and that the moment of intervention of each group must be accurately timed. To ensure perfect co-ordination the spinal reflex arcs and the proprioceptive system regulating tonus must be intact.

It is possible to distinguish three main types of ataxy—the ataxy due to lesions involving the afferent nerves from the muscles and joints, the ataxy due to lesions involving the tonus-controlling cerebellar system, and the ataxy due to lesions of the volitional cortical system.

The first type, or **sensory ataxy**, is always accompanied by some degree of loss of muscle-sense. It is determined by two factors: first, the information conveyed to the cortex as to the position of the limb and the degree of muscular contraction exercised is defective or absent, and hence the effort evoked is not appropriate to the movement to be performed; second, owing to lack of the afferent stimuli the spinal reflex does not enter into play at the right moment or in the right degree. The loss of the sense of position can be to a great extent compensated by visual attention, and hence a patient suffering from sensory ataxy may be extremely ataxic with his eyes shut or in the dark, but when watching his movements may be able to perform them with some degree of accuracy. Thus a tabetic who can-

ATAXY, HEREDITARY CEREBELLAR

not even stand with his eyes shut (*Romberg's symptom*) may walk fairly well when watching his footsteps. Sensory ataxy is the most marked symptom of *tabes dorsalis*; a similar ataxy may occur when the posterior columns of the cord are affected by spinal tumours, by patches of disseminated sclerosis, or in combined degeneration of the posterior and lateral columns. In peripheral neuritis, loss of the afferent sensations from the muscles may persist after the motor fibres have recovered, and give rise to pronounced sensory ataxy.

The second type, **motor ataxy**, is that due to disturbance of the cerebellar system, either of the spino-cerebellar paths, the cerebellum itself, or of its cortical connexions. This form is not accompanied by loss of sensibility, and is therefore not influenced by vision. The cerebellar system is very frequently involved in disseminated sclerosis, and hence ataxy of the motor type is one of the commonest symptoms of this disease.

The third type, **cerebral ataxy**, may arise from toxic affections of the motor cortex, as in alcoholism and extreme fatigue, and occasionally from gross lesions caused by tumours or vascular lesions which have not produced sufficient paralysis to mask the inco-ordination.

Finally, a *spurious ataxy* is frequently a concomitant of hysteria, its diagnosis resting upon its obvious dependence on the attention of the patient to his symptoms and the absence of signs of organic nervous disease.

Treatment.—Sensory ataxy may be treated by re-educational exercises—the patient, if taught to perform slow and ordered movements under the control of vision, as in Frenkel's exercises, may ultimately acquire some knowledge of the degree of cortical effort necessary to perform any given movement (*see also TABES DORSALIS*). Little can be done for non-sensory ataxy in this direction, but when the lesion is stationary the patient acquires to some degree the power of controlling his inco-ordination by bringing accessory fixation muscles into play.

F. L. GOLLA.

ATAXY, HEREDITARY (*see* ATAXY, HEREDITARY CEREBELLAR; FRIEDREICH'S DISEASE).

ATAXY, HEREDITARY CEREBELLAR.

—Marie, who introduced this term, applied it to a group of cases in which the chief symptom was ataxia of movement, of hereditary or familial incidence, which he assumed

ATAXY, HEREDITARY CEREBELLAR

to be due to a primary atrophy of the cerebellum. The symptoms varied considerably in the different families on which he based his description, and subsequent observations have shown that in only one of them was the disease limited to the cerebellum. Hereditary cerebellar ataxia is consequently neither a clinical nor a pathological entity, but the term may be usefully retained as a clinical designation till, by the aid of further knowledge, we are able to separate these cases into types, each with its own characteristic pathological lesions.

Pathology.—In a few of the cases that have been recorded the chief pathological change was a primary parenchymatous atrophy of the cerebellum; in others this organ had been damaged by either acute or progressive changes in the interstitial tissues; some, in which it was the spino-cerebellar tracts that were chiefly affected, may be regarded as anomalous cases of Friedreich's disease; while in many there were diffuse lesions in both the forebrain and the cerebellum. Heredity is rarely observed, but it is common to find two or more cases in the same generation of a family.

The **symptoms** usually appear in early childhood, often as soon as the child begins to walk or use its arms, but the onset is sometimes delayed till the early adult years. The most prominent symptom is irregularity or ataxia of gait; the child who is affected early begins to walk late and never acquires a proper balance, while, if the disease commences later, walking becomes gradually more and more unsteady and assumes a reeling, drunken character. There is usually some ataxia of the arms too; they are awkward and inept in movements and actions that demand accuracy, and are often tremulous. Tremor of the head and trunk is also a common symptom. Speech may become jerky or scanning, but the other bulbar functions are unaffected. Nystagmus is present in some cases, and occasionally there is a squint or other anomaly of the ocular movements. The state of the reflexes varies; when the disease is limited to the cerebellum they remain normal, but the knee-jerks are exaggerated and the plantar responses are of the extensor type when the forebrain or the pyramidal tracts are involved. Sensation is undisturbed. Frequently there are evidences of deficiency or of lack of mental development.

Treatment.—Most forms of the disease are progressive and unrelieved by medical treatment, but carefully selected exercises may

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improve the power of walking. Local lesions of the cerebellum that are amenable to surgical treatment, as tumours and abscesses, must be excluded; they are generally distinguished by the presence of headache, vomiting, and optic neuritis.

GORDON HOLMES.

ATELECTASIS, ACQUIRED (*see* LUNG, (COLLAPSE OF)).

ATELECTASIS, CONGENITAL.—In some cases the lungs fail to expand properly after birth. Occasionally the whole of one lung or lobe, or more usually small patches scattered through the lungs, are found to be non-expanded, retaining the foetal structure. The bases are most commonly affected. The unexpanded areas are depressed and pinkish, and there is no evidence of pleurisy or emphysema. The infant is weak or premature, or there may have been difficulties in labour. The respiratory movements are shallow and feeble, and cyanosis is pronounced, especially in the face and fingers. The skin is cold and clammy, the temperature subnormal, and the pulse weak and irregular. Congenital malformations of the heart may coexist. As might be expected, in the majority of these cases death occurs in a few hours, or at most in a few days. If life is prolonged further, the child remains weak and feeble.

If the symptoms are noticed immediately after birth the **diagnosis** presents little difficulty. Similar symptoms may be seen occasionally in infants with enlargement of the thymus which obstructs the air-passages, but the respiratory movements in such cases are more powerful and noisy.

Treatment.—The child's strength should be preserved by keeping it warm. At the same time an endeavour may be made by general stimulation, smacking, pinching, etc., to cause the child to cry and breathe deeply. These efforts should be persisted in at intervals. In a few cases expansion of the hitherto unexpanded portions takes place.

CHARLTON BRISCOE.

ATHEROMA (*see* ARTERIAL DEGENERATION).

ATHETOSIS AND TREMOR.—The term **athetosis** is in use to signify certain involuntary movements, mostly of the limbs (in particular their distal segments), and also occasionally of the face, which are slow, sinuous, irregular. At their maximum they frequently

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immobilize the limb for a moment or two in a spastic or rather spasmodic attitude, of varying form. At rest, however, the muscles involved are rarely if ever hypertonic; as a rule, indeed, they are mostly hypotonic. The condition is usually unilateral, and is much more commonly facio-brachial or brachial than crural.

Athetosis is more frequently found in association with infantile cerebral hemiplegia than with any other morbid condition; but theoretically there is no reason why athetosis should not occur by itself, i.e. without accompanying involvement of the pyramidal system. It cannot occur if the pyramidal system is severely paralysed; even in pronounced cases, therefore, there is always some voluntary control over the muscles involved. Athetosis, there is little doubt, is the result of lesions on the afferent side of the nervous system, and there is good evidence to suggest that it is the sequel to impairment of function of that afferent system which, arising in the dentate nucleus of the cerebellum, passes by the superior cerebellar peduncle to the red nucleus of the opposite side, thence to the optic thalamus, and so to the sensory-motor cortex.

So-called *double athetosis* is a disease belonging to the group of cerebral diplegias (see CEREBRAL DIPLEGIA), but the actual symptom of athetosis occurring in it is identical with what has been above described.

Treatment.—No nerve sedatives have any enduring or even transient effect on the involuntary movements. If they are slight, the patient himself gradually learns to ignore them, or at least regards them with equanimity. If they are severe, the only treatment that can be of any value is surgical. Horsley removed the corresponding motor cortex in one case, and thus, by paralysing the muscles concerned, checked the athetosis. Unfortunately for the patient, the question of athetosis or paralysis remains a sort of Hobson's choice.

Tremor is defined as an involuntary movement consisting in the alternating innervation of any given muscular group and its antagonists; hence results a more or less rhythmical, regular, to-and-fro displacement of a limb or segment of a limb. The amplitude or excursion of the limb or segment involved is, naturally, variable, and so is the rate of oscillations per second. Some tremors are fine and quick, others are slow and wide. They may be of a pendulum-like regularity, or highly irregular. They may increase with voluntary movement

AURICULAR FIBRILLATION

(action- or intention-tremor), or this may inhibit their activity. Sometimes they disappear when the limb is supported at the corresponding joints, and appear only when the limb is held out.

The number of diseases of which tremor in one or other of its forms is a manifestation is notably large, and only the most important of them can be enumerated.

Of organic conditions, paralysis agitans, disseminated sclerosis, progressive lenticular degeneration, and exophthalmic goitre may be mentioned. Tremor occurs in certain toxic and toxi-infective conditions, e.g. lead-poisoning. It is common in the neuroses (hysteria, neurasthenia, etc.). There is evidence to show that tremor depends for its external manifestation on impairment of function of the efferent system from the corpus striatum, through the red nucleus to the spinal cord via the rubro-spinal tract. It seems fairly clear, too, that lesions of the cerebello-rubral system are in some instances associated with tremor, more particularly of the intention type. Hence any lesions of the midbrain (tumours, softenings, etc.) may have tremor as one of the symptoms. So-called familial and idiopathic tremors may also be mentioned.

Treatment.—As in the case of athetosis, efforts to diminish or check the involuntary movement by means of nerve sedatives are unsatisfactory. At one time or another bromides, hyoscyamus, scopolamine, formates, opium, etc., have been tried without noteworthy results. In every case the practitioner should aim at ascertaining the underlying pathological condition, and treating it along approved lines. With success in this direction, the tremor, as one of the symptoms of the disease, may be expected to improve along with other symptoms. In only too many instances, however, it remains peculiarly intractable.

S. A. KINNIE WILSON.

ATRESIA VAGINÆ (see AMENORRHOEA).

AURICULAR FIBRILLATION.—An abnormal rhythm of the heart in which the auricles fail to contract and remain distended in fibrillary tremor while the ventricles beat in a completely irregular fashion.

The name is applied clinically to those cases of myocardial disease in which auricular fibrillation, and consequently a total irregularity of the pulse, is a dominant and usually a permanent feature.

AURICULAR FIBRILLATION

Etiology.—Acute rheumatism is by far the commonest general infection causing the myocardial lesion which later reveals itself in auricular fibrillation. The arrhythmia rarely appears during the course of the infection itself, and, like the mitral stenosis which so often accompanies it, must be regarded as a sequel. Diphtheria, scarlet fever, enteric fever, influenza, and, of course, chorea are occasionally causative.

The degenerative changes in the heart due to age, in some cases combined with arteriosclerosis or chronic renal disease, are the commonest cause in the elderly. It is not uncommon in advanced stages of exophthalmic goitre and during the course of septic endocarditis.

In a patient predisposed by infection or by senile changes a sudden physical strain may determine its onset, though strain alone in a healthy subject is not an adequate cause for auricular fibrillation.

Pathology.—The post-mortem findings in cases which have shown fibrillation during life have not yet revealed the exact pathological foundation for its occurrence. Fibrosis of the heart-muscle, especially of the auricular, is a common but not an invariable association; this change is often present without an abnormal rhythm. Mitral stenosis is found as an accompaniment very frequently, and aortic incompetence occasionally.

Briefly stated, the current view of the mechanism of this rhythm is that the minute lesions left widely in the auricular muscle by acute rheumatism or other infection progress until, some years later, they suddenly begin to produce multiple irritative stimuli. These set the auricular wall into a state of fibrillary tremor and supersede the normal impulses arising from the sino-auricular node, which is the pace-maker of the auricle and therefore of the heart. The ventricle now receives from the auricle such rapid and indiscriminate impulses that its rhythm cannot be other than completely irregular. The additional circulatory embarrassment is due to this rapid and

irregular ventricular beat rather than to the absence of auricular systole.

A more recent hypothesis proposes that depressed conduction in the auricular muscle leads to a breaking-up of the excitation wave. This spreads widely and evokes fibrillary contractions in the auricular wall—fibrillation.

Symptomatology.—Auricular fibrillation commonly arises between the ages of 20–50 when the rheumatic cases predominate, and after 50 when the influence of age enters into play. It is more frequent in men than women, and notably rare in childhood. The onset is usually evidenced by a sudden accession of symptoms of heart failure in a patient who has previously suffered little from a latent myocardial lesion or from recognized valvular disease like mitral stenosis. When the new rate proves to be relatively slow, especially in the elderly, the onset of symptoms is by no means so abrupt, for they may only appear gradually some time after the inception of the new rhythm. Often a few paroxysms of fibrillation lasting for a few hours or days precede its permanent establishment by a period of weeks or months. When it has become permanent, as it almost invariably does, the normal limits of cardiac response are restricted and the patient remains incapable of severe or prolonged exertion, though he may enjoy fair health for years. He may be regarded as more or less an invalid with heart disease, or at the very least as one who is more prone than others to heart failure. Undue exertion, i.e. exertion beyond the restricted limit imposed on the heart by the new rhythm, provokes breathlessness and pain. With advancing heart failure, orthopnoea, cyanosis, dropsy, and enlargement of the liver supervene.

The cardinal feature presented on examination is the complete irregularity of the heart, noticeable on palpation and auscultation as well as on feeling the pulse (Fig. 6). It is present whenever the patient is examined, and is accentuated by slight exertion. The rate in untreated cases is usually 90–180 per minute, though in a few it never becomes



Fig. 6.—Auricular fibrillation. Radial tracing, showing complete irregularity at a high rate, 160–170 a minute.

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high. The apex beat is commonly felt beyond the nipple line, while other signs denoting enlargement may be obtained by percussion or by radioscopy. If mitral stenosis had been indicated by a presystolic murmur or thrill when the heart was regular, it may be inferred with equal certainty during fibrillation from a *diastolic* murmur or thrill limited to the apex. Clearly a presystolic murmur cannot be expected when auricular systole has ceased to occur, and its replacement by one in diastole, just after the second sound, usually becomes obvious when the ventricle is slowed by digitalis. Systolic murmurs in all areas are common, and offer little guidance or help in any direction. Aortic incompetence is far less frequent than mitral stenosis in association with fibrillation; but it should not be missed, for it presents its own characteristic sign—an aortic diastolic murmur well heard in the third left space, with the irregularity of fibrillation superimposed.

To sum up, the symptoms of auricular fibrillation are identical with those of heart failure, often urgent at its onset and depending in degree largely upon the rate of the ventricle. The signs are the signs of heart failure accompanied by a completely irregular pulse.

Diagnosis.—The frequency of auricular fibrillation may be realized from the fact that the majority of patients admitted to a general hospital with heart failure and dropsy are examples of this condition. It may be diagnosed when symptoms of heart failure are present if the pulse is completely irregular at a rate over 100 and the irregularity persists indefinitely. With a rate of 60–100 the irregularity is not so striking; but exertion (such as raising the arms a few times), by increasing the rate, will render it unmistakable, besides dispersing two irregularities which may simulate it at the lower rates. These are:

(1) *Sinus arrhythmia*.—This is normal at any age, and may be observed in most children, especially when the pulse is slow. It consists of a sequence of quickening during inspiration and slowing during expiration; it is exaggerated by deep respirations, and disappears if the subject stops breathing.

(2) *Premature contractions (extrasystoles)*.—These are seldom so numerous and persistent as to simulate the irregularity of fibrillation. They are dissipated on raising the pulse-rate as by slight brisk exertion, and have not the serious significance of fibrillation.

If there remains a doubt as to the differential diagnosis of the irregularity, it will be settled by a graphic record (*see HEART-BEAT, ABNORMALITIES OF*). The polygraph and electrocardiograph are only necessary for a practical diagnosis in a small proportion of cases, but they are decisive and final. In both methods the diagnostic features of the record are (i) the total irregularity of the ventricular beat, and (ii) absence of the wave *a* or electrical variation *P* denoting auricular systole (Fig. 7). More detailed information and greater certainty are often obtained from an electrocardiogram than from a polygram. This is especially true of a third condition to be distinguished from fibrillation, namely, flutter.

(3) *Auricular flutter*.—This abnormal rhythm is much less common than fibrillation but similar in many clinical particulars and in that it is an evidence of myocardial disease. When at rest the subject of flutter may exhibit a perfectly regular pulse, sometimes only 75 a minute, more often about 150. Irregularity difficult to distinguish from that of fibrillation occurs on movement or during digitalis medication. In any case, it is not easy to diagnose without an electrocardiogram.

Prognosis.—The presence of auricular fibrillation in a case of valvular or myocardial disease at once renders the prognosis more serious. Not only is it a sign of myocardial involvement, but the frequency and irregularity of the ventricular action constitute an additional burden on the heart which is soon manifested by the increasing severity of the symptoms. From the point of view of prognosis it must not, however, be considered apart from the other signs, for in exceptional cases its effect seems slight, while in others it rapidly precipitates a complete breakdown of the circulation. Apart from the symptoms induced, the index upon which most reliance may be placed is the *rate* of the heart, best counted by auscultation when a polygram is not available. In general terms the prognosis is grave in patients showing a high ventricular rate, such as 120–180 or perhaps uncountable, which fails to fall and then to remain at a moderate rate such as 60–80 under an adequate course of digitalis. In such cases it will be found that the symptoms as well as the pulse-rates are badly controlled by digitalis. Extreme enlargement of the heart is a serious sign. Cases of aortic incompetence do not as a rule improve under treatment so well as cases of mitral stenosis. If paroxysms of

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fibrillation occur they are usually a prelude to the permanent condition. Any of the various complications of valvular disease, such as cerebral embolus or septic endocarditis, may of course intervene with this abnormal rhythm present. Better results from treatment may be anticipated in young adults with rheumatic lesions than in elderly patients with arterial and myocardial degeneration, though the latter may show few symptoms for years if the pulse-rate is slow, as is sometimes the case even without digitalis. The outlook in the average case is not gloomy, and the

that its action may almost be regarded as specific. Without delay, tincture of digitalis (B.P.) should be administered in doses of 20 min. or more, three times a day (1-1½ dr. daily). Its action is best followed when it is prescribed in simple solution without unworthy and confusing adjuvants. In two or three days symptoms should begin to abate, urine to increase, and the pulse-rate to fall. While the improvement is maintained, this dosage is continued until the pulse-rate falls to about 70, when the digitalis should be reduced to 10 or 15 min. t.d.s., or withheld for a few

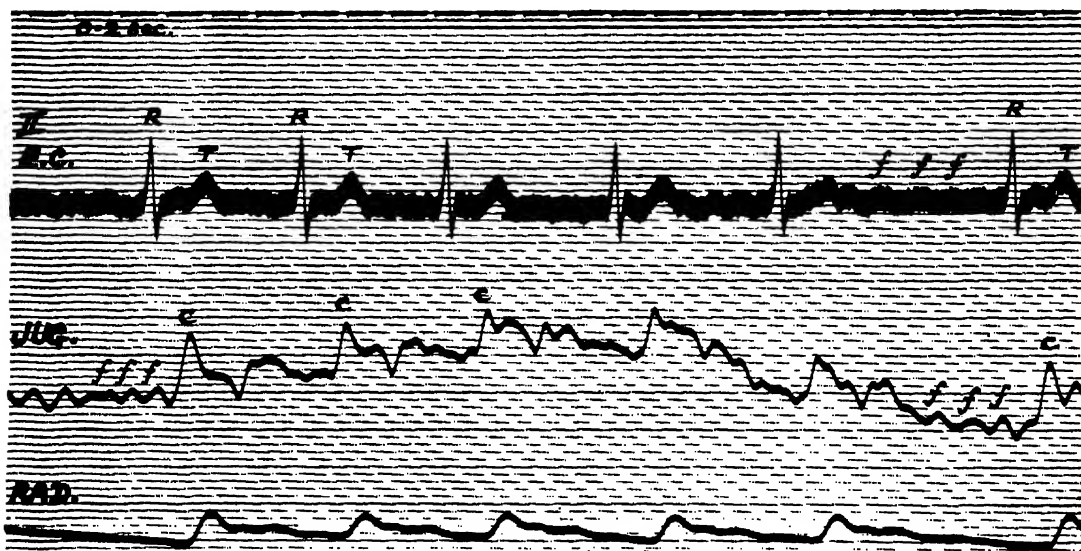


Fig. 7.—Auricular fibrillation. Triple record of electrocardiogram with jugular and radial tracings. The waves P and a of auricular contraction are absent, and small fibrillation waves (f) are visible. The ventricular contractions (R, c, and radial beats) are irregular, and at a slow rate (60-70 a minute) under the influence of digitalis.

subject of auricular fibrillation may continue for months—more likely, indeed, for years—quite able to do light work or, at least, to remain tolerably free from severe symptoms when refraining from work.

Treatment.—Acute heart failure, whether precipitated by the onset of fibrillation or occurring in its chronic course, is treated by rest and digitalis. Confinement to bed with a dry diet in small meals is advisable while the pulse-rate is high, or while dropsy and other urgent symptoms are present. Digitalis cannot cure auricular fibrillation or remove the complete irregularity of the pulse, but it possesses such power of reducing ventricular rate in this condition and improving the symptoms

days (not more) and then resumed at the lower dose. Slight nausea or headache does not call for its cessation, and a single vomit may be disregarded if merely incidental; but persistent vomiting, severe headache, a pulse-rate below 60, and coupling of the beats are the signs of digitalis-poisoning and indicate that the drug must be stopped forthwith.

If a patient is first seen in extreme straits from heart failure with fibrillation and a high pulse-rate, an intravenous injection of 1/200 to 1/100 gr. of strophanthin may be used, provided always that digitalis has not been given, as it is dangerous in a patient already under the influence of this drug. It may be repeated in twenty-four hours, or digitalis may then

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be used by mouth in its stead. Reduction in pulse-rate and improvement of symptoms often follow within an hour.

Venesection to the extent of 20-30 oz. affords relief in cases with grossly distended veins and pulsating liver, though seldom for more than a day or two.

There is an important exception to the rule that digitalis is indicated in auricular fibrillation. If short paroxysms appear for the first time in a patient and lead to no alarming symptoms, digitalis should be withheld, because these attacks sometimes pass away spontaneously, not to return again for months. It is a curious fact that the administration of this drug in the paroxysmal form of the disease tends to make the fibrillation permanent. Apart from this exception, digitalis medication may be superfluous if the rate is naturally slow and the symptoms are trivial.

When the severer symptoms have subsided and the patient can get about, there are two principles which govern his treatment. (1) The daily life must be adjusted on a lower scale of exertion to correspond with the limited heart force under the permanent handicap of auricular fibrillation. The amount of exertion permissible may be of any degree which stops short of producing distress. Light employment and walking exercise on the level can usually be undertaken, but not manual labour of any strenuous character. (2) The optimum dose of digitalis for continued administration should be found by trial in co-operation with the patient. The improvement in symptoms and increased capacity for exertion under its influence are generally so plain to the patient that he will not do without it. There is no danger from its continued administration if the signals given above are regarded. The time is past when a patient should be allowed to relapse time after time when so often a small dose of digitalis regularly taken will save him from heart failure or indefinitely postpone it. An average dose for such continued use is 10-15 min. t.d.s., though 5 min. will sometimes suffice. The optimum dose will be that which prevents complications such as dropsy, permits reasonable exertion without much discomfort, and maintains a moderate pulse-rate of 60-80 a minute.

JOHN PARKINSON.

AURICULAR FLUTTER.—A special form of paroxysmal tachycardia in which the auricular rate is so high, about 300 beats a minute, that the ventricle seldom if ever responds to

each auricular impulse, but contracts at a lower rate, often a half, the disparity of rates depending upon the degree of heart-block.

In its tendency to become permanent and in its serious significance, flutter is akin rather to auricular fibrillation than to the ordinary form of occasional paroxysmal tachycardia.

Etiology.—An acute infection in which the myocardium has been affected may some years later result in the appearance of auricular flutter. The commonest of these infections is acute rheumatism; diphtheria, influenza, and syphilis are other causative infections. From these and from acute and chronic infections of unknown origin arise most of the cases in patients below the age of 40.

Apart from the infective, a rather larger degenerative group is encountered among patients showing signs of advancing years, in general arterio-sclerosis and degenerative changes in the coronary arteries and the heart-muscle. Individual cases definitely traceable to an infection may only develop flutter when they have reached an age where senile changes form an additional or even a determining factor in its production. More than half the recorded cases of flutter have been over the age of 40, and the majority have been men. Among less common causes may be mentioned food-poisoning, chloroform-poisoning, and hyperthyroidism. Flutter may be a terminal event preceding and accelerating death from heart failure.

Pathology.—The exact pathological foundation of auricular flutter is as doubtful as that of auricular fibrillation. The acute myocarditis of an acute infection, chronic myocardial fibrosis, and degenerative changes associated with coronary disease are some of the conditions which have been found in the necropsies in cases in which during life flutter occurred; sometimes no myocardial change has been found. Acute and chronic pericarditis or endocarditis are naturally met with in association, and also the congestive changes in the liver and other organs associated with heart failure.

Experimentally, flutter can be readily produced in the heart of a dog by stimulation of the auricle with a weak faradic current; in fact, flutter was described as a physiological phenomenon long before it was recognised clinically.

The present conception of auricular flutter is that the abnormal excitation wave, once started, travels perpetually in the auricle. Delayed conduction in the auricular muscle

AURICULAR FLUTTER

allows one portion to return to rest and become non-refractory before the slowly-spreading impulse re-enters it. Hence it contracts again, and the process is repeated indefinitely—the "circus movement." The associated heart-block is due to the fact that the ventricles are unable to follow the auricles at such extreme rates, so that every second auricular beat or so fails to produce a ventricular response. The extreme rapidity of the ventricle, when it does temporarily follow each auricular beat, is probably responsible for the syncopal attacks sometimes seen in flutter.

Symptomatology.—(1) In patients with apparently healthy hearts, flutter may occur as short paroxysms from time to time without the same symptoms and course as ordinary paroxysmal tachycardia. Without apparent or adequate reason the tachycardia suddenly begins with a sensation of rapid beating in the neck and chest and a stifling feeling rising to the throat. The subject finds himself breathless after climbing a few stairs, and weak on accustomed exertion. Fullness and discomfort below the left breast are common, and aching in this region may persist for a day or two after the attack, but severe pain is rare. All the symptoms are aggravated by exertion—indeed during recumbency little may be felt. Yet prolongation of the paroxysm, even in a patient at rest in bed, may not only maintain but increase breathlessness and other symptoms and actually lead to graver indications of cardiac failure, such as orthopnoea, cyanosis, swelling of the liver, and oedema. With the sudden termination of the attack the symptoms abate, and in a few hours the patient may feel as fit as before it occurred. Between the paroxysms serious symptoms are absent, though there is often sufficient limitation of the heart's reserve to make the patient short-winded.

(2) In patients with the signs and symptoms of heart disease, flutter is a serious complication, whether occurring in paroxysms or in the permanent condition to which these tend, or as an intermediate stage in the genesis of permanent fibrillation. It leads to an aggravation of the symptoms which accompanied the heart disease. The new and abnormal rhythm compels a heart already showing signs of disease to beat at an excessive rate and so to exhaust its force.

The patient with heart disease who develops flutter may experience symptoms identical with those described under (1), as occurring in a paroxysm. But from that time he shows

symptoms more readily, his attacks recur more frequently, and even between them the complaints become more insistent. When flutter has become permanently installed the patient is prone to heart failure, and the diminution of his heart force is sufficient to limit or prevent manual work. He complains of breathlessness on exertion, of palpitation persistent or noticeable only on movement, of fullness and aching pain in the left submammary region of pressure in the epigastrium, of a suffocating feeling in the throat, and of similar conditions arising from a heart in a violent hurry. On the other hand, some patients show great tolerance of the flutter, and continue for months or even years with few symptoms. In them it can be assumed that the heart-muscle is not extensively affected by disease, and can better bear the tachycardia.

In certain cases of flutter, though by no means in all, there is a special tendency to feelings of faintness, and even to true syncopal attacks, which add to the gravity of the outlook.

The physical signs are variable, and often give no clue to the underlying abnormal rhythm. The symptoms and signs which should suggest it are described under diagnosis. Often nothing abnormal will be found by ordinary clinical examination. In those who are already the subjects of heart disease the appropriate signs will be observed, in some respects modified by the new rhythm. Thus the murmurs of mitral stenosis, evident enough with a normal rhythm, may be obscured in its presence. The heart-sounds may have the characters of embryocardia. The position of the apex beat varies with the individual case, and should always be noted because of its importance in prognosis. In course of time serious symptoms and signs tend to appear, e.g. orthopnoea, cyanosis, enlargement of the liver, and oedema.

Diagnosis.—As auricular flutter can only be diagnosed with certainty by instrumental methods, it is all the more important that the symptoms and signs which suggest its presence should be remembered.

Any form of palpitation may be described by a patient as "flutter"; and a fluttering pulsation observed in the neck may be occasioned by forms of tachycardia other than auricular flutter.

An adult who develops suddenly or within a few days the symptoms of heart failure such as severe breathlessness, with or without a

AURICULAR FLUTTER

previously recognized cardiac lesion, is probably the victim of an abnormal rhythm, either ordinary paroxysmal tachycardia or else auricular fibrillation or flutter. It is presumed that non-cardiac causes such as pneumonia or renal disease have been excluded, and that no signs of acute carditis are present.

(1) Ordinary *paroxysmal tachycardia* (q.v.) often recurs at intervals of weeks or months as a troublesome palpitation of typically sudden onset and offset; but the pulse is always regular and often uncountably frequent. It is distinguished from flutter by the form of the electrocardiogram, and by the absence of block.

(2) The clinical difference between *auricular fibrillation* and flutter is slight; the causes, the incapacity, the tendency to permanence are similar; and flutter often ends in fibrillation.

the block is 2:1 the interpretation is often uncertain, and other tachycardias can show similar jugular tracing. The radial pulse may show pulsus alternans when the ventricular rate is high (Fig. 8).

The electrocardiograph is undoubtedly the proper instrument for the diagnosis of auricular flutter. The auricular (*P*) waves are as steady and as readily counted as the graphic record of a vibrating tuning-fork. Superimposed are seen the higher ventricular (*R*) waves after every second, third, or fourth *P* wave, or at irregular intervals if the block is varying (Fig. 9).

Prognosis.—Occasionally flutter runs a course like ordinary paroxysmal tachycardia, in that attacks occur from time to time causing inconvenience or distress, but symptoms are

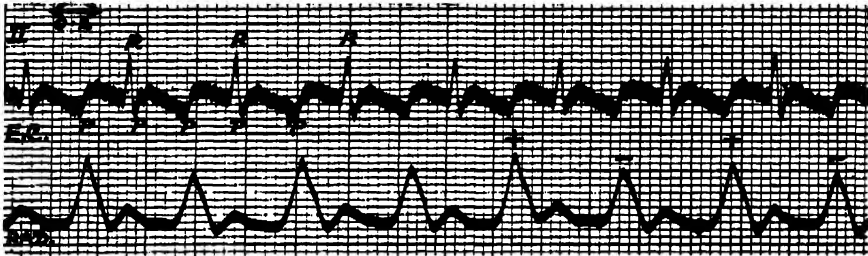


Fig. 8.—Auricular flutter. Electrocardiogram with radial tracing. The auricular contractions (*P*) are regular at a rate of 300 a minute. The ventricular contractions (*R*) follow every alternate auricular beat (*P*) at a rate of 150 (2:1 block). The radial tracing is regular at 150 a minute, and shows pulsus alternans.

A useful diagnostic point is that the pulse is always irregular in fibrillation while it is most often regular (and rapid) in flutter. Another difference lies in the fact that while the irregular pulse of fibrillation is labile and will increase on exertion, the regular rate of flutter is fixed as in all forms of regular paroxysmal tachycardia. It is true that the radial pulse in a case of flutter is sometimes very irregular at an occasional examination because of a varying block often induced by movement or by digitalis; but it is more characteristic to find the pulse regular at about the rate of 150 with 2:1 block (Fig. 8), or about 75 with 4:1 block, or even 150 at one moment and 75 at another.

A definite diagnosis can sometimes be made by means of the polygraph. In the jugular tracing is seen a rapid succession of auricular (*a*) waves at 250–300 a minute, and the carotid and radial waves appear in response to every second, third, or fourth auricular beat. Where

slight or absent between the attacks, and signs of heart disease do not develop. A prognosis should therefore not be essayed in a first attack.

More often its onset is an event of importance as indicating the existence of myocardial disease and, where this is already obvious, as a factor in emphasizing the symptoms and accelerating heart failure. Attacks of flutter, like those of fibrillation, tend to recur and to give place to the permanent condition of flutter, and this quite often ends in permanent fibrillation. Flutter is a serious complication if only because it compels the burdened heart to contract at a very high rate, the auricle—and, more important, the ventricle—being robbed of sufficient diastolic rest. Permanent flutter, as permanent fibrillation, almost always connotes limitation of the heart's functional power, even if it does not produce actual failure for a long time. A weighty factor in prognosis is the rate of the ventricle. If there

AURICULAR FLUTTER

is naturally much block, e.g. 4 : 1, so that the ventricular rate is about 75, it is clear that this is preferable to a block of only 2 : 1 with consequent pulse-rate of about 150. In default of such a natural block, it is promising if this optimum ventricular rate of 75 or so can be maintained by the block imposed by digitalis, where this drug has failed to dissipate the abnormal rhythm altogether.

Great enlargement of the heart is as usual unfavourable, and cases presenting frequent syncopal attacks are always in peril. It will be seen that a reliable prognosis in flutter

dosage, is nothing less than disappearance of the flutter and return to the normal rhythm. This is effected in a large proportion though scarcely in the majority of cases, and the remarkable sequence under digitalis is often—flutter, increasing block, temporary fibrillation, and return to the normal rhythm. The patient may then remain "cured" for years, though sometimes only for months, after which the flutter is likely to return.

Even if this full object is not attained, the results from digitalis are often surprisingly

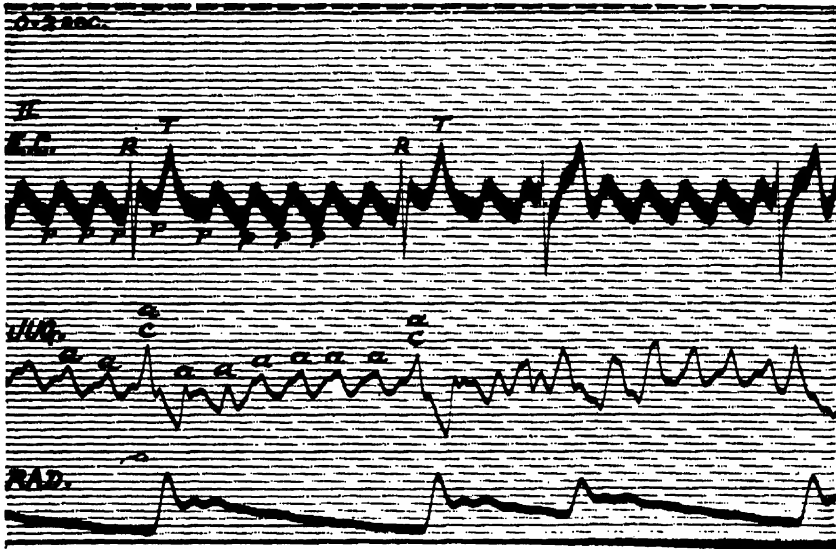


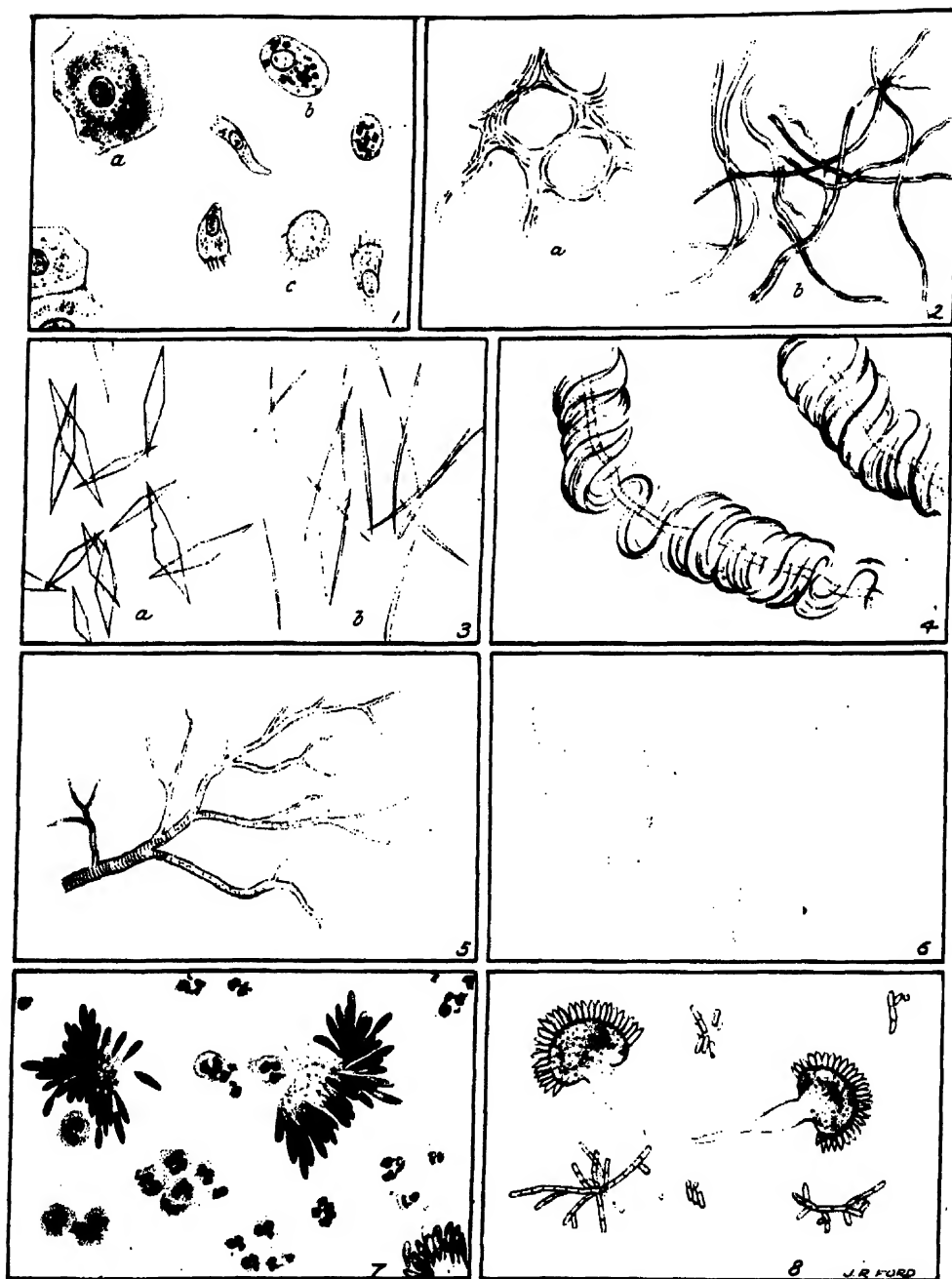
Fig. 9.—Auricular flutter. Triple record of electrocardiogram with jugular and radial tracings. The auricular contractions (P and a) are regular at 240 a minute. The ventricular contractions (R and c and radial beats) are irregular and slow, the result of varying block induced by digitalis.

cannot be given without taking into account all the associated factors, and of these the most useful are the ventricular rate, the reaction to digitalis, and the observed effect (as shown by his symptoms) which the new rhythm produces on the particular patient.

Treatment.—For patients without the symptoms and signs of heart disease, rest in bed is all that is required during the short and occasional attacks of auricular flutter, and digitalis should be avoided.

Where attacks are prolonged, so that physical signs of failure result, or where the flutter has become permanent, digitalis generally proves of remarkable benefit if given freely. The optimum result, often obtained only by large

good. For it is not so much the high auricular rate which tries the heart as the consequent acceleration in the rate of the ventricle. By increasing the block, digitalis produces ventricular slowing, and with a ventricular rate restrained to 70–80 a minute the symptoms and signs almost always abate, the auricular flutter notwithstanding. But the dose of digitalis must be adequate; at least a drachm of the official tincture daily will be necessary to meet the need of the average patient, until the pulse-rate is reduced to 70–80 the symptoms are eased, and the signs begin to disappear. Then and not till then should the daily ration be reduced. Slight headache and slight nausea, or even an isolated vomit, ought not to inter-



1, Epithelium, $\times 350$: (a) stratified; (b) pulmonary; (c) ciliated. 2, (a) Elastic tissue, $\times 50$; (b) elastic fibres, $\times 350$. 3, (a) Charcot-Leyden crystals, $\times 300$; (b) fatty acid crystals, $\times 300$. 4, Curschmann's spirals, $\times 10$. 5, Bronchial cast, $\times 10$. 6, Fibrin casts, $\times 10$. 7, Actinomyces, $\times 400$. 8, *Aspergillus fumigatus*, $\times 350$.

PLATE 3.—SUBSTANCES FOUND IN SPUTUM.

BACTERIOLOGY AND PATHOLOGY, CLINICAL

rupt the medication upon which so much depends. But severe headaches, repeated vomiting, coupled beats, and a pulse-rate below 60 are the four danger signals any one of which calls for a stoppage of the drug. Irregularity of the pulse with flutter merely means a varying block instead of an even 2:1, 3:1, or 4:1 ratio, and has no bearing on the treatment. It is the ventricular rate itself, whether regular or irregular, which is the essential guide to dosage. It may be counted with advantage by auscultation at the apex during a whole minute.

If a patient with flutter is first seen in extreme heart failure, and digitalis has *not* been given, a more rapid result may be obtained by a single intravenous injection of strophanthin, gr. 1/200-1/100, obtainable conveniently dissolved in ampoules. This may be repeated after twenty-four hours, or digitalis itself may

then be begun. Similar prompt treatment is demanded by the recurrence of syncope attacks which threaten life.

When the happy result of digitalis-therapy has been a reversion to the normal rhythm, the drug should be eschewed and the life of the patient so ordered as to avoid any distress and strain which might provoke a return of the flutter. When the result has been simply a reduction in the ventricular rate and an improvement in the symptoms, digitalis should be continued *indefinitely* in a dose, say 15-45 min. of the tincture daily, sufficient to keep the rate well below 100. The patient will feel at his best under these conditions, always provided he avoids any exertion which is found to induce distress.

JOHN PARKINSON.

AUTUMNAL CATARRH (see HAY FEVER).

BAOILLARY DYSENTERY (see DYSENTERY).

BACILLURIA (see CYSTITIS; PYELONEPHRITIS AND PYELITIS).

BACTERIOLOGY AND PATHOLOGY, CLINICAL.—This subject will be considered under the headings of—

1. EXAMINATION OF SPUTUM.
2. THROAT AND NOSE SWABS.
3. CLINICAL EXAMINATION OF THE BLOOD.
4. BLOOD-CULTURES.
5. EXAMINATION OF CEREBRO-SPINAL FLUID.
6. EXAMINATION OF EXUDATES.
7. EXAMINATION OF CYST-CONTENTS.

1. **Examination of sputum.**—In examining sputum it is most important to work on suitable material. If the patient is intelligent, the best plan is to provide him with a sterile bottle and to instruct him to spit into it when he feels that a mass of sputum has come directly from the lungs. Failing this, it is best to use the early morning sputum, and if necessary the precaution should be taken of telling the patient to clean his teeth and wash out his mouth overnight; by this means the presence of particles of food, which interfere

much with the examination, can to a great extent be avoided. The presence of large amounts of saliva and of secretion from the naso-pharynx often renders a proper examination of sputum very difficult and the result misleading.

As the first step towards the examination, it is advisable that the sputum should be spread on a Petri dish or a sheet of glass (this must be sterile if cultures have to be made) and examined carefully with the naked eye and with a low-power lens. By this means particles of pus or of muco-pus can be distinguished from the more watery saliva and a favourable specimen may be selected for examination; further, Curschmann's spirals, scraps of elastic tissue, and other unusual materials may be picked out. (PLATE 3.)

The examination usually required is two-fold, microscopical and cultural. For the *microscopical* examination select a characteristic piece of sputum about the size of a half-pea, remove it with a platinum loop, and place it on the centre of a clean microscopical slide. Take a second slide and press the two firmly together so as to flatten the sputum out into a thin film; in doing so hold the two slides crossing one another very obliquely, almost parallel with one another in fact; then slide the two apart, taking care not to lift one from

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the other as you do so. This will give you two good films. There may be thick masses at the edge of the films, but these you can remove with a platinum loop or piece of blotting-paper. Allow the films to dry, and stain one by Gram's method, and one for tubercle bacilli. First fix them in the flame by passing them three times, rather slowly, through the flame of a Bunsen burner, so that the backs of the slides get so hot as to be slightly uncomfortable to the back of the hand.

(1) **Gram's method.**—Gram's solution is made from (a) saturated alcoholic solution of gentian violet and (b) aniline oil water. Aniline oil water is prepared by shaking a few cubic centimetres of aniline oil in about 100 c.c. of distilled water for ten minutes or so. The solution thus obtained is filtered through two layers of filter paper, previously moistened with distilled water, and must be quite clear before use. To prepare the stain, take 9 parts of this solution and 1 part of the alcoholic solution of gentian violet. As the stain does not keep very well, it is important that the practitioner should know how to prepare it. Another requisite is Gram's iodine solution, consisting of 1 grm. of iodine, 2 grm. of iodide of potassium, and 300 c.c. of water. To prepare it, place the iodine and iodide of potassium in a bottle, add a small amount of water, 3 or 4 c.c., and shake. Then add the rest of the water. If it is attempted to dissolve the ingredients in the whole amount of water, the iodine will be found to dissolve with extreme slowness.

The following are the stages of the method—

(a) Stain the fixed film in Gram's solution for three minutes or so, then pour away the stain and wash for a few seconds in water.

(b) Flood the film with iodine solution and allow it to act for a minute. Pour the fluid away and replace with a second dose, which should also be allowed to act for a minute.

(c) Decolorize with absolute alcohol or with strong methylated spirit until no more colour comes out. Rinse rapidly in water to remove the alcohol.

(d) Counterstain for about half a minute with a $\frac{1}{2}$ -per-cent. solution of neutral red in water, or for ten seconds with carbol-fuchsin diluted with ten times its volume of water.

(e) Lastly, wash the film thoroughly, blot with fluffless blotting-paper, dry, and examine under the oil-immersion lens. It will be found that some micro-organisms are stained very deep violet, almost black, and others red.

This is a very important factor in the recognition of micro-organisms generally, and especially of those found in sputum.

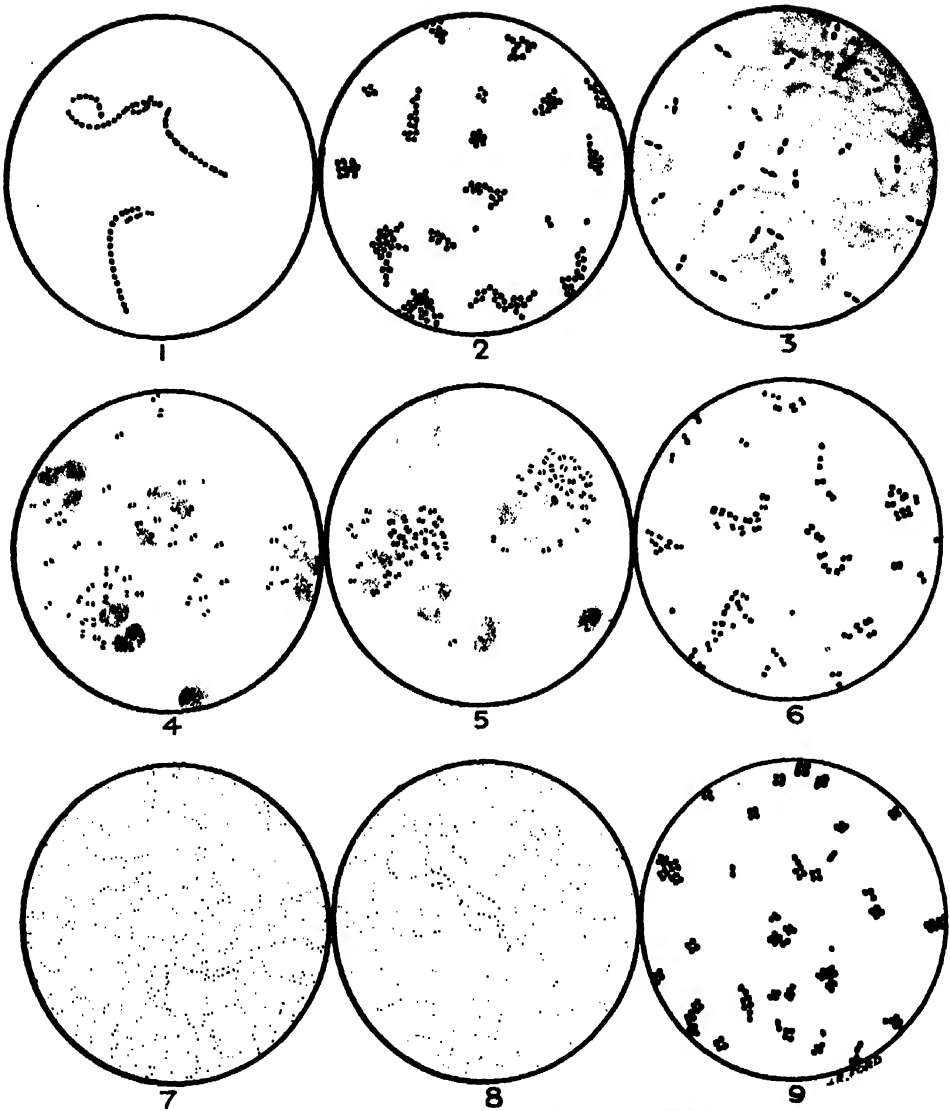
CLASSIFICATION OF THE CHIEF PATHOGENIC ORGANISMS ACCORDING TO THEIR POWER OF RETAINING GRAM'S STAIN. (PLATES 4, 5)

<i>Gram positive</i>	<i>Gram negative</i>
COCCI	
Streptococcus	Meningococcus
Staphylococcus	Gonococcus
Pneumococcus	M. catarrhalis
M. tetragenus and M. paratetragenus	M. melitensis.
BACILLI	
B. anthracis	The typhoid-coli group, including the dysentery bacilli, etc.
B. diphtheriæ	B. influenzae
B. tuberculosis *	B. mallei
B. lepræ *	B. pestis
B. tetanus	B. fusiformis (bacillus of Vincent's angina).
B. edematis maligni	
B. perfringens	
MISCELLANEOUS	
Actinomyces	Vibrio cholerae
Ringworm and other pathogenic fungi	Spirilla or spirochaetes of relapsing fever, syphilis, and Vincent's angina, and all pathogenic protozoa.
Yeasts	

* Are "acid-fast" also.

(2) **Staining for tubercle bacilli (Ziehl-Neelsen's method).**—Carbol-fuchsin is prepared by adding 1 part of a saturated solution of basic fuchsin in alcohol to 9 parts of 1-in-20 solution of carbolic acid in water. This has to be used with gentle heat; the simplest way is to boil the stain in a test-tube and pour it whilst boiling on to the fixed film. Or the slide may be supported, film side upwards, on a suitable support, flooded with carbol-fuchsin, and a spirit-lamp applied until steam begins to rise. The slide should be stained in the hot carbol-fuchsin for not less than five minutes.

Next rinse off the stain, and decolorize by immersing the slide bodily in sulphuric acid—the exact strength does not matter, but anything between 5 and 25 per cent. will do. When decolorization is complete the colour will not return, or, if it does, only as a faint tinge when the slide is washed under the tap. If in these circumstances much colour remains, re-immerses the slide in the acid. This stage of the pro-



1, Streptococcus. 2, Staphylococcus. 3, Pneumococcus. 4, Meningococcus. 5, Gonococcus.
6, Micrococcus catarrhalis. 7, M. melitensis. 8, M. paramelitensis. 9, M. tetragenus.

PLATE 4.—PATHOGENIC MICROCOCCI.
(Stained with Gram and counterstained with neutral red.)

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cedure can hardly be overdone, and the tubercle bacilli, if properly stained at first, will not be decolorized if left in the acid for an hour or more.

After washing the slide, counterstain it with a dilute solution of methylene-blue; any preparation will serve. Wash, blot, dry, and examine as before.

By this method of preparation only the tubercle bacilli, the leprosy bacilli and the smegma bacilli are stained red; other microbes are stained red in the first part of the process, but lose their colour when immersed in the acid, and are finally stained by the methylene-blue. Certain micro-organisms occurring in butter, milk, etc., are stained red by this process, but their presence in sputum is so unlikely that the value of the method is not thereby impaired.

2. **Throat and nose swabs.**—These are required chiefly for the detection of the diphtheria bacillus and of the meningococcus. They may also be needed for the diagnosis of Vincent's angina.

(1) **Diphtheria.**—A swab may be required either for the diagnosis of the disease or for determining whether the subject is a "carrier," either after or in the absence of an attack. To collect it the patient should be put in a good light and the swab pressed firmly against any lesion that may be present, or, if there is no lesion, rubbed firmly on both tonsils. If there is a false membrane, a small portion should be withdrawn on the swab, if possible.

The swab should be inoculated on blood serum, as this is a very suitable medium for the diphtheria bacillus, whereas most other organisms grow on it much more slowly. The blood-serum culture medium is very easily prepared if sterile or chloroformed horse-serum is available, and, as this is now an ordinary article of commerce, the method will be given.

Place about 5 or 6 c.c. of serum in a clean sterile test-tube, put this in a suitable serum inspissator and raise the temperature to 70° C. Keep it at this temperature for thirty minutes, and you will find that the serum has coagulated, forming a white, firm mass. Meat extract or concentrated broth may be added to this, but is not necessary, nor for the diagnosis of diphtheria is it advisable. When the culture medium is not at hand, the swabs may be sent

for a considerable distance, as the diphtheria bacillus is not an organism which dies readily outside the body. The cultures may be examined after twelve hours. The diphtheria bacillus will have made a considerable amount of growth, whereas most other organisms will have made very little. The colonies of the diphtheria bacilli are small, white, and raised, and such colonies should be picked out by a platinum loop and emulsified in a small drop of water on a clean slide, which is then dried, fixed by heat, and stained. *Methylene-blue* is one of the finest stains, and the following formula (*Manson's stain*) can be recommended: Methylene-blue 2 gm., borax 5 gm., water to 100 c.c. This solution keeps well, and should be diluted with 5-10 times its volume of water before use. Now wash, blot, and dry, and examine with an oil-immersion lens.

It is often advisable to make the diagnosis as quickly as possible. The characters of the diphtheria bacilli of the throat exudate are not quite the same as in cultures, and the organisms are less distinctive. If, however, a membrane is present and a part of the lower surface of this can be rubbed on a slide, the presence of large numbers of Gram-positive bacilli is practically conclusive.

The diphtheria bacillus can be recognized by these simple methods with a fair degree of certainty. Difficulties arise occasionally, however, which can only be settled with certainty by animal inoculations.

(2) **Cerebro-spinal meningitis.**—The organisms can most easily be found in the postnasal space, where sometimes they are present in practically pure culture. To collect them the most convenient method is to use a *West's swab* (Fig. 10), consisting of a clean tube, curved round through a quarter circle at one end, so as to pass readily behind the soft palate. Inside this there is a

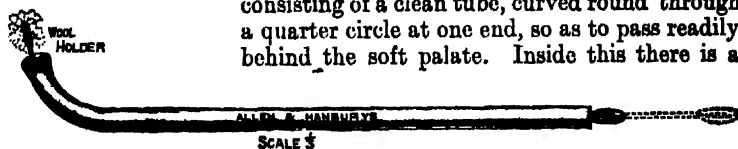


Fig. 10.—West's swab.

swab made of flexible wire, which is withdrawn when the swab is passed, so as not to be contaminated with the saliva, etc. It is not always easy to pass it on a sensitive patient. A tongue-depressor is often necessary, and the tip of the glass tube should be inserted sideways under the soft palate, and then rotated in the middle line. When the tube is in position, push out the swab so that it projects beyond the end of the tube, and move it

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gently from side to side at the back of the nasopharynx. Then withdraw the swab into the tube. The further examination is entirely cultural, and it is necessary to use special media consisting of agar that contains, in addition to its usual ingredients, blood, serum, or some other of the numerous ingredients which recent research has discovered. This is pre-eminently a matter for the expert, for two reasons. (a) The organism dies very readily, and the inoculation and incubation of the culture medium must be made within half an hour or less of collecting the swab. (b) The identification of the meningococcus from the throat is rendered difficult by the presence of non-pathogenic organisms which very closely resemble it, and in many cases are only differentiated from it by rather complex serological tests.

(3) **Vincent's angina.**—This is a not uncommon affection, but it is often mistaken for diphtheria or syphilis. It is important that it should be recognized, since it is readily curable by simple means. Collect the specimens as before and spread them on slides; stain one of these by methylene-blue, as directed for diphtheria, and another by Gram's stain, as recommended for sputum. In the methylene-blue specimen numerous long bacilli, pointed at each end and often containing one or more vacuoles, will be seen, and also long spirilla with a few open curves. The latter usually stain rather badly, and may appear very faint with this stain. Neither of the organisms stains by Gram, and will appear of a red colour. It is a good plan also to examine a wet preparation of the material between slide and cover-glass, when both organisms will be seen in rapid movement, and usually present in vast numbers.

3. Clinical examination of the blood.—The ordinary clinical examination of the blood should include (1) Estimation of hæmoglobin, (2) Enumeration of red corpuscles, (3) Study of the morphology of red corpuscles, (4) Enumeration of leucocytes, (5) Study of the morphology of leucocytes.

(1) **Estimation of hæmoglobin.**—The only really accurate method by which this can be accomplished is that described by Newcomer, which involves the use of a colorimeter and a glass colour-standard. But for clinical purposes, Haldane's and Sahli's methods are convenient, fairly accurate, and in common use.

Haldane's method (Fig. 11).—Place a little water in the graduated tube, prick the patient, and suck the blood up to the 20-c.mm. mark on

the pipette. Wipe the tip of the pipette free from blood and lower it into the bottom of the water in the graduated tube. Blow gently to expel the blood, when it will form a layer at the bottom of the vessel. Raise the tip slightly and suck up water, and again expel it. This may be repeated two or three times, and in this way every trace of blood can be washed out of the pipette. Now shake so as to mix the blood and the water together. The hæmoglobin (which is liberated from the cor-

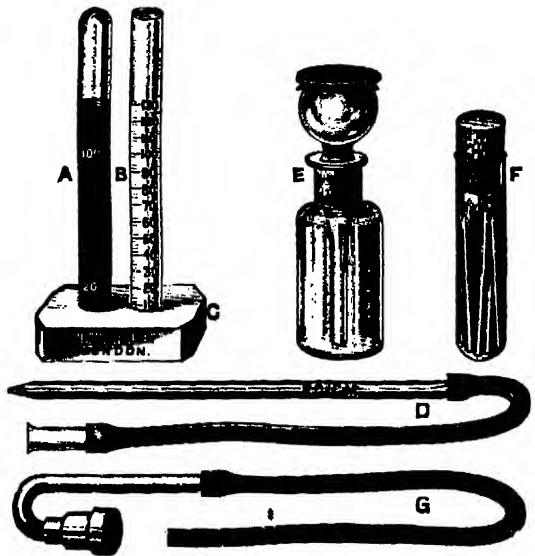
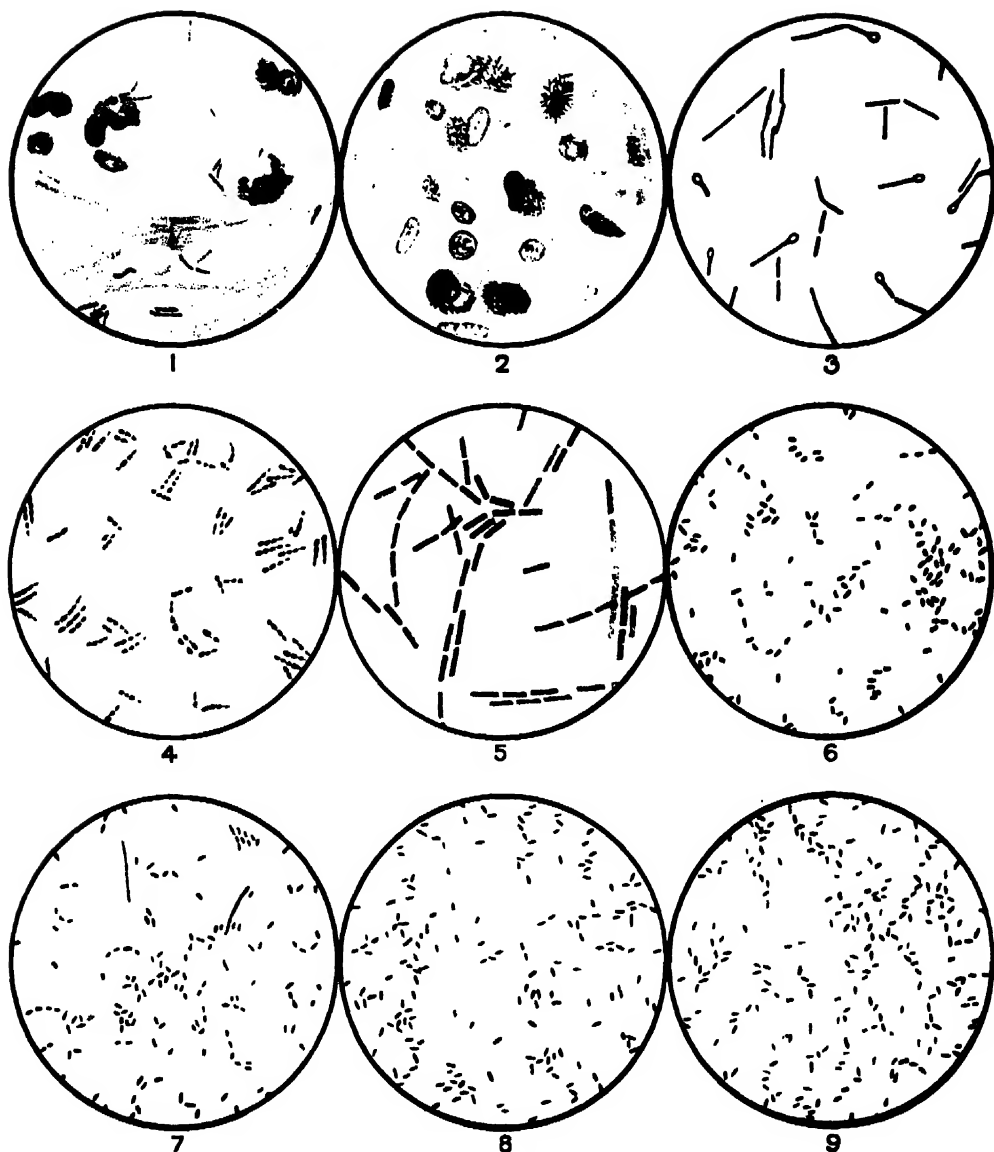


Fig. 11.—Haldane's hæmoglobinometer.

A, Standard blood solution; B, graduated mixing tube; C, rubber stand; D, pipette; E, bottle for distilled water; F, lancets; G, tube and cup for fixation to gas-burner.

puscles as they are "laked" by the water) has now to be converted into CO-hæmoglobin by filling the tube with coal gas, stoppering the end with the finger, and shaking gently. This should be repeated two or three times, when an obvious change in colour will take place in the solution. A curved tube which will fit on to an ordinary gas burner is supplied with the apparatus for the purpose of conducting gas into the tube. The solution in the graduated tube is now compared with that in the sealed standard tube. It should be darker, and if this is not the case, too much water was taken at the outset, and in these circumstances the simplest plan is to take an additional 20 c.c. of blood and proceed as before, in which case it will be necessary to halve the result ultimately obtained. Now add water drop by drop, shaking after each addition, until the



1, *Bacillus tuberculosis* in sputum (Ziehl-Neelsen). 2, *B. lepræ* in skin (Ziehl-Neelsen). 3, *B. tetani*, some showing spores (Gram-staining, not counterstained). 4, *B. diphtheriæ* (methylene-blue). 5, *B. anthracis*, not showing spores (methylene-blue). 6, *B. typhosus* (Gram and neutral red). 7, *B. paratyphosus*-A. 8, *B. paratyphosus*-B. 9, *B. paratyphosus*-C.

PLATE 5.—PATHOGENIC BACILLI.



colour exactly matches that in the standard tube. The amount of hæmoglobin is now read off directly in percentages on the graduated tube.

Sahl's hæmoglobinometer.—The process is exactly the same except that the dilution is made in 1 per cent. hydrochloric acid in water. This hæmolyses the blood and converts the hæmoglobin into acid hæmatin, and the amount of hæmoglobin is read off by comparison with a similar solution sealed in a standard tube the diluted blood being further diluted until the two colours just match. The standards for these hæmoglobinometers should be kept in a dark place, as they gradually fade.

(2) **Enumeration of red corpuscles.**—The apparatus most used for this purpose is the Thoma hæmacytometer (Fig. 12). This consists of a diluting pipette in which a 1-in-100 dilution of the patient's blood in some isotonic fluid can rapidly be prepared, and the counting chamber in which the actual enumeration takes place. The usual dilution fluid is Toison's, which is made up as follows:—

Distilled water . .	30 c.c.
Sodium sulphate . .	8 grm.
Sodium chloride . .	1 grm.
Methyl violet . . .	A trace.

The method is as follows: Having pricked the patient, suck the blood into the lower limb of the hæmacytometer pipette until it just reaches to the line marked 1 (that nearest the bulb), and then place the tip in the diluting fluid and suck gently, rotating the bulb of the pipette as you do so, so that the two fluids are rapidly mixed and coagulation has not time to occur. Suck until the mixture extends to the line marked above the bulb, and

away the Toison's fluid remaining in the lower part of the stem. Then put a small drop of the diluted blood in the middle of the counting-chamber and gently lower the cover-glass into position, taking care not to include an air-bubble. The cover-glass should be firmly pressed into position by pressure with a pair of needles, or similar objects, on the four corners in succession, and Newton's rings should appear where pressure is applied; this shows that there is actual contact between the cover-glass and the surface on which it rests. (A better plan is to clip down the cover-glass with four Cornet's forceps applied at the corners, or with the clips which are supplied with some recent instruments.) Allow the corpuscles to settle for five minutes or so and then proceed to count.

On examination, the central part of the inner disc will be seen to be divided into small squares, 400 in number, and forming a chess-board 20 squares each way. These squares are $\frac{1}{10}$ mm. across, and, as the space between the glass on which they are ruled and the cover-glass is $\frac{1}{10}$ mm. in depth, it follows that the cubic capacity of the fluid immediately over each is $\frac{1}{1000}$ c.mm. The corpuscles in this bulk of the fluid will settle down on to the space below it, so that by finding the average number of corpuscles on each square and multiplying by 4,000 we obtain the number of corpuscles in one cubic millimetre of the fluid. Inasmuch as the blood is already diluted 100 times, it will be necessary to multiply this number by 100 to obtain the number in 1 c.mm. of undiluted blood. The simplest way to obtain this average is as follows: Count five "bars" of the squares, one at the top of the chess-board, one at the bottom, and three intermediate. Begin at one side of the large square, and count the whole 20 squares in the bar, moving the counting chamber gradually across the field so as to keep the area you are counting in the middle. Any corpuscles which lie on the upper line of the bar, or on the left-hand side, should be counted as within the bar, while those which are lying on the right-hand side or the lower margin should not be counted; this will ensure your counting each corpuscle once, and once only. These five bars will give you 100 squares; the total number should be added up, and this number divided by 100 will give you the average.

(3) **Morphology of red corpuscles (PLATE 6).**—Of the methods which have been suggested for preparing films for examining the morphology

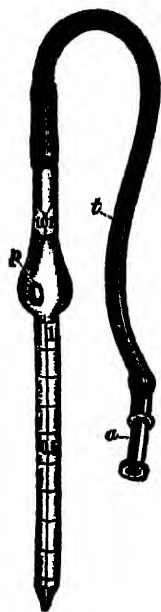


Fig. 12.
Thoma hæmacytometer.

then, closing each end of the pipette with the finger and thumb, give another good shake. The fluid in the bulb will now be a 1-in-100 dilution of the blood.

Next take the counting-chamber and clean it and the thick cover-glass with great care, using a soft linen handkerchief, which you should keep for the purpose. Blow out about one-third of the fluid in the bulb so as to wash

of red corpuscles and leucocytes, by far the best is that involving the use of two cover-glasses: methods by which the blood is smeared into an elongated film on a slide, though not without their uses, are to be deprecated. Use $\frac{1}{2}$ -in.-square cover-glasses (not too thin, or they will buckle in the process of spreading the film); have them scrupulously clean and polished with a soft linen rag. Take one cover-glass between the thumb and finger of the left hand and hold the other by the adjacent corners with the thumb and finger of the right hand. Now take a small drop of blood (the size can only be determined by experience) on the centre of the upper surface of the cover-glass held in the left hand, and place the centre of the other cover-glass in contact therewith, holding both cover-glasses horizontal. Now let go of the upper cover-glass, and if you have taken the right amount of blood you will find that it will spread into a uniform film just filling the opposing surfaces of the two cover-glasses. When the film has ceased to spread, take hold of the upper cover-glass by the corners you have just relinquished, and slide the two rapidly apart. This should give you excellent films, which will present a uniform distribution of red corpuscles and leucocytes throughout.

The best methods of staining are Jenner's and Leishman's. The former presents some advantages in simple blood work, but as Leishman's method (or one of its modifications) is essential for working with some of the protozoa, it is probably best to use this method throughout.

(a) *Jenner's method*.—Take a cover-glass, film side up, between a pair of Cornet's forceps and flood the surface with the stain. Allow it to act from one to four minutes (the exact length of time necessary for efficient staining for any particular specimen of Jenner's stain can only be learnt by experience), then wash rapidly in distilled water for about a quarter of a minute and blot the film between two layers of fluffless blotting-paper or filter-paper, taking care not to wipe it. Dry rapidly by gentle warmth over a flame and mount in balsam.

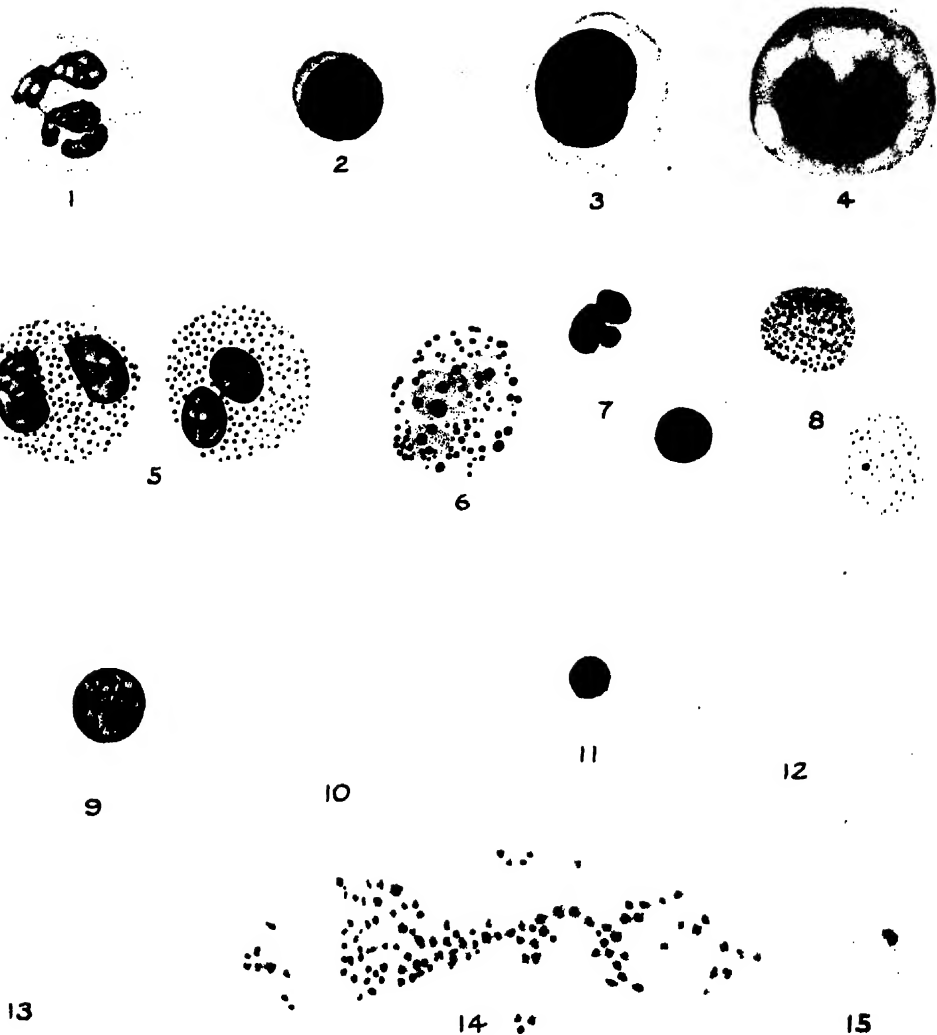
(b) *Leishman's method*.—The technique is the same as for Jenner's, except that, after the stain has acted for some two minutes, about the same amount of distilled water has to be mixed with it and the staining process allowed to proceed for another two or three minutes.

In Jenner's method the active ingredients are eosin and methylene-blue in chemically

equivalent preparations. These are dissolved in methyl alcohol, which acts as a fixing agent, so that no previous fixation is necessary. The methylene-blue stains all the nuclei, though with different degrees of intensity, and the granules of some cells. The eosin stains the red corpuscles a pink colour, the depth of which is approximately proportional to the amount of the hæmoglobin they contain. It also stains the granules of the eosinophil corpuscles, and, to a less extent, those of the polynuclears. Leishman's stain has a similar action, but here there is a third ingredient which stains the chromatin substance of protozoa, e.g. the malaria parasite, bright red. The nuclei of the red corpuscles are stained purplish with this stain, but except for this its action is very much like that of Jenner's.

(4) *Enumeration of leucocytes*.—The simplest method is to utilize the preparation already employed for counting the red corpuscles; no special pipette is used. The leucocytes being scanty in proportion to the red corpuscles, it is necessary to count a much larger volume of the mixture (1 in 100) than suffices for the latter. To aid this, numerous "field methods" have been devised, of which the following has been in constant use in my laboratory for many years, and has been found highly convenient. Arrange the microscope in such a way that the edge of the field is tangential to two lines of the counting-chamber which are eight bars apart. This involves the use of a $\frac{1}{4}$ -in. objective and a No. 2 eye-piece. The draw-tube of the microscope is now drawn out until the requisite magnification is obtained.

(5) *Differential leucocyte count*.—The stained film which was prepared for the morphology of the red blood-corpuscles is used. This is examined under a $\frac{1}{2}$ - or $\frac{1}{4}$ -in. objective, using a mechanical stage. Beginning at the one corner of the preparation, the slide is moved across the field of the microscope, while the observer notes down, by means of an initial, the different sorts of leucocytes (PLATE 6) as they are seen, distinguishing polynuclears as P., lymphocytes as L., etc. It will be found convenient to adopt some method for registering these numbers, so that one can add them up quickly, or know at any time how many have been counted. A good plan is to put them down in blocks of 25, or in rows of 10 each. To get a really correct count, not less than 400 or 500 should be counted, but for many clinical purposes 250 is often enough. Having examined the requisite number, add the numbers



J.R.FORD, *sen.*

1, Polynuclear leucocyte. 2, 3, Small and large lymphocytes. 4, Large mononuclear (large hyalin). 5, Eosinophils. 6, Mast-cell. 7, Normoblasts. 8, Punctate basophils, one showing polychromasia. 9, Megaloblast. 10, Megalocyte. 11, Microcyte. 12, Poikilocytes. 13, Red corpuscles showing polychromasia. 14, Blood-platelets. 15, Blood-platelet lying on a red blood-corpuscle.

PLATE 6.—BLOOD CELLS, NORMAL AND ABNORMAL.
(Leishman's Stain.)

1. The first part of the document is a list of names and addresses of the members of the committee.

2. The second part is a list of the names of the members of the committee.

3. The third part is a list of the names of the members of the committee.

4. The fourth part is a list of the names of the members of the committee.

5. The fifth part is a list of the names of the members of the committee.

6. The sixth part is a list of the names of the members of the committee.

7. The seventh part is a list of the names of the members of the committee.

8. The eighth part is a list of the names of the members of the committee.

9. The ninth part is a list of the names of the members of the committee.

10. The tenth part is a list of the names of the members of the committee.

11. The eleventh part is a list of the names of the members of the committee.

12. The twelfth part is a list of the names of the members of the committee.

13. The thirteenth part is a list of the names of the members of the committee.

14. The fourteenth part is a list of the names of the members of the committee.

15. The fifteenth part is a list of the names of the members of the committee.

16. The sixteenth part is a list of the names of the members of the committee.

17. The seventeenth part is a list of the names of the members of the committee.

18. The eighteenth part is a list of the names of the members of the committee.

19. The nineteenth part is a list of the names of the members of the committee.

20. The twentieth part is a list of the names of the members of the committee.

21. The twenty-first part is a list of the names of the members of the committee.

22. The twenty-second part is a list of the names of the members of the committee.

23. The twenty-third part is a list of the names of the members of the committee.

24. The twenty-fourth part is a list of the names of the members of the committee.

25. The twenty-fifth part is a list of the names of the members of the committee.

26. The twenty-sixth part is a list of the names of the members of the committee.

27. The twenty-seventh part is a list of the names of the members of the committee.

28. The twenty-eighth part is a list of the names of the members of the committee.

29. The twenty-ninth part is a list of the names of the members of the committee.

30. The thirtieth part is a list of the names of the members of the committee.

31. The thirty-first part is a list of the names of the members of the committee.

32. The thirty-second part is a list of the names of the members of the committee.

33. The thirty-third part is a list of the names of the members of the committee.

34. The thirty-fourth part is a list of the names of the members of the committee.

BACTERIOLOGY AND PATHOLOGY, CLINICAL

of the different sorts of leucocytes present and reduce them to a percentage.

4. Blood-cultures.—For the examination of blood for the ordinary cultivable micro-organisms a considerable quantity is necessary, as they are often present in very small numbers. As a rule, not less than 10 c.c. should be used. If possible, it should be collected about the time when the patient's temperature is at its maximum, as it is found by experience that the chances of success are greater at these periods than at others. The blood, as soon as it is collected, should be mixed with a considerable quantity of sterile broth; for every 10 c.c. not less than 100 c.c. of broth should be used, and twice this amount is an advantage. If the blood is diluted with a small amount of fluid, the bacterial substances which are present may be sufficient to destroy the organisms—e.g. those of the typhoid group. A special culture medium may be used which inhibits the action of these bacterial substances considerably—e.g. bile in the case of typhoid bacilli and its allies. Another method is to add trypsin to the broth; this probably has a double action, adding amino-acids and other nutrients, and also inhibiting or destroying antibacterial substances. One c.c. of any good glycerin solution of trypsin should be added to the flasks of broth with a sterile pipette and the latter incubated for twenty-four hours before the medium is required for use. The blood is collected by venepuncture, a sterile needle attached to a sterile syringe or pipette being used. I greatly prefer a pipette holding about 15 c.c. and ground to fit the needle; the other end, which is constricted, is plugged with cotton-wool and the pipette enclosed in a large test-tube also plugged with cotton-wool; the whole is sterilized by dry heat. This ensures its absolute sterility, and the whole apparatus may be kept indefinitely, ready for immediate use as required. If a syringe is used it should be of the all-glass variety, with an accurately fitting piston, and should be sterilized (in a metal box) in an autoclave. The use of a syringe is very much to be deprecated, as no suction is required to extricate the blood (and, indeed, suction, by drawing the wall of the vein against the point of the needle, may defeat its own object), and the exposure to the air, necessary in fitting the parts of the syringe together, is a fruitful source of contamination.

Technique of venepuncture.—Place a tourniquet consisting of an india-rubber tube, clipped by a pair of Spencer Wells forceps, round the

upper arm tightly enough to impede the venous flow but not so tightly as to interfere with that in the arteries. Get the patient to clench his fist or, preferably, to squeeze a suitable object such as a roll of bandage. Now examine the antecubital fossa and select a suitable vein for puncture, one that is firm and elastic, and does not slip easily from side to side before the finger; it matters not that it is deep so long as the firm elasticity can be felt. Sterilize the skin thoroughly with tincture of iodine. Now take the pipette or syringe and push it obliquely through the skin, about half an inch from the spot which you decide to be the most suitable for puncture, keeping the needle in the line of the vein; it is immaterial whether it is pointed in the direction of the blood-stream or against it. When the vein is entered the blood will rise quickly in the pipette. If the syringe is used, a little gentle suction may be required in order to overcome the friction of the piston, but in a well-fitting syringe this should not be necessary. Allow the blood to rise until the requisite amount has been collected, then release the tourniquet, withdraw the needle, and quickly expel the blood into the broth, by pressure of the piston if the syringe is used, or by blowing through the further end of the pipette. Give the flask of broth a rotary shake to ensure rapid mixing, and put it in the incubator as soon as possible.

Some bleeding may occur after the withdrawal of the needle; this can be stopped by firm pressure with a wedge of cotton-wool, or will cease spontaneously if the patient's arm is raised. A small piece of cotton-wool wetted with collodion may be used to seal the puncture, but the use of a bandage is not advised, because of the increased risk of thrombosis in the punctured vein.

The culture.—As soon as the blood has been withdrawn, and before it has had time to clot it should be mixed with the broth. The practitioner may, if he chooses, inoculate solid culture media or liquefied agar with a series of sterile pipettes, but this is not recommended, as any chance of air-borne contamination should be reduced to its minimum. After twenty-four hours' incubation the culture should be examined with the naked eye. The presence of a diffuse turbidity, or of hæmoglobin dissolved in the broth, is a probable indication of growth, and the culture should be examined forthwith. Using a sterile pipette, withdraw some of the fluid, and immediately

inoculate one or two agar tubes. Submit what remains in the pipette to a microscopical examination. Of course, if there are obvious colonies, some of these should be withdrawn in the pipette; in any case it is better to take the deposit rather than the supernatant fluid. Allow the flask to be opened for as short a time as possible, and return it to the incubator immediately. Blood-cultures should not be considered negative until four days have elapsed, as sometimes there are in the blood a few organisms which have been so acted on by the defensive substances there as to be barely viable; these may not grow in the cultures for three or four days.

5. Examination of cerebro-spinal fluid.

—The amount of cerebro-spinal fluid necessary for a full examination is not more than 5 c.c., and in ordinary circumstances less may be taken, so as to avoid "puncture headaches." (For the technique of the operation, see LUMBAR PUNCTURE.) It is advisable to collect the fluid in two tubes, because the first may be contaminated with a few red blood-corpuscles, which would interfere with the enumeration of the leucocytes; the second tube, which should be entirely free from red corpuscles, is to be used for this purpose. If the fluid is under pressure it is well to go on until this is approximately normal.

Technique.—This will depend on the nature of the disease that is suspected. In *acute meningitis* (cerebro-spinal fever, tuberculous meningitis, etc.) the bacterial examination is all-important. Inoculate tubes of appropriate culture media from the first tube, and centrifugalize the rest, making films suitable for staining from the deposit. From the second tube take a small amount of the fluid and place it on a Thoma counting-chamber, just as is done with diluted blood when a blood-count is made. Cover it, allow the corpuscles to settle, and proceed to count the number of leucocytes, using the field method (p. 150). If you count 80 fields you will have the number of leucocytes without further calculation; usually 40 are sufficient, and in this case you have simply to multiply by 2 to get the number per cubic millimetre. The second tube should be allowed to clot, as the method of coagulation will often give important information. The fluid from tuberculous meningitis, for instance, clots slowly, the clot having a fairly characteristic spider-web appearance, and when the tube is gently shaken it retracts from the side and forms a slender thread,

which, if removed and examined, will often reveal the presence of tubercle bacilli when examination of a centrifugalized deposit has proved negative. If this has been done the supernatant fluid should be treated chemically. In health there is a mere trace of albumin and sugar present. In meningitis the albumin is greatly increased and sugar is often absent. In tuberculous meningitis, however, the sugar, though reduced, is often present in small amount, and the deposit of cuprous oxide formed on testing with Fehling's solution may take some hours to appear.

When *syphilitic* or *parasyphilitic* infections are in question the important examinations are—

(1) Estimation of the number and nature of the cells.

(2) Wassermann reaction.

(3) Globulin test.

(4) Colloidal gold test.

This may also be regarded as the order of importance.

(1) The method for estimating the *number* of cells has been described already. Their *nature* may be recognized by the experienced eye while this examination is being made, but to make sure it is advisable to stain a film of the centrifugalized deposit. In health there are not more than one or two cells per cubic millimetre, whereas in syphilitic affections the number may be increased to 100 or more. The cells are practically all lymphocytes.

(2) For the *Wassermann reaction*, see under SEROLOGICAL DIAGNOSIS.

(3) *The globulin test.*—The amount of globulin in the cerebro-spinal fluid is usually increased in parasyphilitic affections and in syphilitic meningitis. There are several tests for this, of which only the simplest can here be given. Take a small (measured) amount of a saturated solution of ammonium sulphate (about 1 c.c.) in a narrow test-tube and float on it an equal quantity of the fluid under examination. The presence of a whitish ring at the junction of the two fluids, readily visible to the eye, indicates an increase of globulin. After the fluids have been in contact for a few minutes they should be gently shaken and the effect read off a few minutes afterwards. Normal cerebro-spinal fluid gives a very slight opalescence, whereas in syphilitic affections of the nervous system the mixture may become very decidedly turbid. Experience with normal cerebro-spinal fluid should be obtained, if possible, before too much reliance is placed on this test.

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CHARACTERS OF THE CEREBRO-SPINAL FLUID IN HEALTH AND DISEASE

	COLOUR	TRANSPARENCY	CELLS	PROTEIN	SUGAR	COAGULATION	PRESSURE	WASSERMANN	GLOBULIN REACTION	ORGANISMS
<i>Normal</i> .	Colourless	Perfect	One or two per c.mm.	Trace	Trace about 0.1%	Absent; sometimes a few fibrils after some hours	Normal	Negative	Absent	Nil
<i>Meningitis: Cerebro-spinal</i>	White or greyish, may be faint yellow or greenish	Turbid or opaque	Numerous polynuclears	+	Normal, diminished, or absent	Takes place	+	—	+	Meningococcus
"Septic"	"	"	"	+	"	"	+	—	+	Various
<i>Tuberculous</i>	Colourless	Slightly impaired	Numerous lymphocytes	+	Diminished or absent	Characteristic spider-web	+	—	+ or —	Tubercle bacilli
<i>Syphilis: General paralysis</i>	"	Perfect	Lymphocytes 50-200 per c.mm.	Slightly increased	Normal	Normal	Normal or slightly +	Strong	+	Nil
<i>Tabes</i> .	"	"	" (often fewer)	"	"	"	Normal	Usually present, rarely strong	+ (usually)	"
<i>Uremia*</i> .	"	"	Sometimes an excess of lymphocytes	Normal, or may be +	"	Normal, or may resemble that of tuberculous meningitis	Often +	—	Absent	"
<i>Diabetes</i> .	"	"	Normal	Normal	+	Normal	Normal	—	—	"

* The important diagnostic feature of the cerebro-spinal fluid in uremia is the presence in excess of urea (more than 0.04 %) and of chlorides (more than 0.42 %).

(4) *The colloidal gold test.*—If the meninges are involved, cerebro-spinal fluid precipitates colloidal gold from an emulsion, and this is claimed by some to be a method by which syphilitic meningitis, tabes, and general paralysis can be differentiated. The test is a difficult one to carry out; for details special works should be consulted.

Presence of blood in the cerebro-spinal fluid.—Blood may occur in the cerebro-spinal fluid from a hæmorrhage in the brain, cord, meninges, pareties, etc., or from injury by the needle in its passage through the pareties; or the bevel of the needle may lie partly in a small vein and partly in the spinal canal, so that a mixture of recently effused blood and cerebro-spinal fluid may escape. It is in the highest degree important to differentiate these conditions, for the actual presence of red blood-corpuscles in the fluid whilst in the body indicates hæmorrhage and is of considerable diagnostic value.

Blood drawn during the operation of lumbar puncture is not, as a rule, equally mixed with the fluid; it may be absent or slight in the last portion of the fluid withdrawn and abundant in the first, or vice versa. The leucocytes and red corpuscles, and the various kinds of the former, are present in the same proportions as those in which they occur in the circulating blood; the fluid is, in fact, exactly similar to the patient's blood mixed with saline. Lastly, it clots, though this process may take place slowly and imperfectly.

When the blood comes from a hæmorrhage, especially from a deep hæmorrhage of the brain, or from any lesion that has lasted a few days, these characters, as a rule, are profoundly changed. The fluid rarely clots; if it does so the hæmorrhage is very recent, or the fluid has an admixture of blood drawn during the puncture. The blood and the fluid are thoroughly mixed, and there is no difference in the amount in the first and last portions. Further, a few hours after the hæmorrhage has taken place there is an excess of leucocytes, and especially of the polynuclears; and after a few days *macrophages*—large cells having the power of ingesting red corpuscles, which they often contain in large numbers—may make their appearance. Lastly, as the blood disappears the clear fluid becomes of a yellow colour. These criteria will usually enable a diagnosis to be made with certainty.

6. Examination of exudates (pleuritic fluid, etc.).—Important information as to the

nature of morbid exudates may often be obtained from an examination of the bacteriology and of the cytology of the fluid. Its chemistry is of less importance, but sometimes information may be obtained from a chemical examination also (*see* PATHOLOGY, CHEMICAL). In many cases the fluids undergo clotting after withdrawal from the body, an event which interferes greatly with the subsequent microscopical examinations. This clotting may be avoided by adding to the specimen a small amount of citrate of potash or soda, as soon as it has been withdrawn; and the practitioner is advised to take with him, when he is about to make such an exploratory puncture, a tube containing a small amount ($\frac{1}{2}$ c.c. or so) of a saturated solution of one of these reagents. He should also take culture tubes and inoculate them on the spot, thus avoiding any possibility of contamination through the tube containing the citrate, which should be used for the microscopical examination only.

The method of examination will depend to a large extent on the appearance of the fluid and its supposed cause. Thus clear, or almost clear, fluid will suggest tubercle, renal disease, etc., whilst a turbid fluid will point to an acute infection by the pneumococcus or streptococcus. In the former case the cytological examination is the more important; in the latter, cultures and the microscopical search for organisms.

If the exudate is clear, or almost clear, centrifuge it, or allow it to settle, decant off the clear supernatant fluid, and examine some of the deposit, wet, between a slide and a cover-glass, with a $\frac{1}{4}$ -in. objective. You can readily recognize lymphocytes as small round cells; they usually occur in abundance in tuberculous exudates. Endothelial cells are much larger and have large nuclei; sometimes they are rounded, sometimes irregular in shape, and fitting into one another so as to form small sheets; the latter have desquamated directly from the endothelium lining the serous cavity, while the former have proliferated off from endothelium remaining *in situ*. The presence of these cells in considerable numbers is suggestive of renal disease, or of passive exudates due to back-pressure. Some polynuclear cells may also be recognized: if in small numbers their significance may not be great; if abundant they suggest an early empyema, and bacteriological and cultural examination of the fluid becomes important.

The cytological examination may be facilitated by adding a small drop of methylene-

blue at one side of the cover-glass and allowing it to soak in. This will colour the cells and render their nature more recognizable.

Another film should also be stained from the deposit according to the methods described for sputum (p. 146). It may be pointed out, further, that tubercle bacilli are usually very scanty in these exudates and can only be found after a prolonged examination, if at all. Whenever tuberculosis is suspected, the only certain way of making the diagnosis from an exudate is by inoculating some of the deposit into a guinea-pig; in view of the great importance of early diagnosis in such cases, this is often advisable. Turbid fluid generally indicates infection by pneumococci or streptococci; empyema due to other organisms may occur, but is uncommon. Tuberculous empyemata are not uncommon, and in them the tubercle bacilli may usually be detected more easily than in the clear exudates. The cultures taken at the time of the operation should be incubated for twenty-four hours and then examined. Films should also be made in the same way as recommended for blood-cultures (p. 151), or, if the material is thick and purulent, in the way recommended for sputum. The nature of the infection can usually be ascertained by microscopical examination without much difficulty. Do not forget that the prognosis in pneumococcal empyemata is the better the fewer the organisms present, and that this is especially the case if they are found mostly outside the polynuclear leucocytes, which form the bulk of the cells making up pus. Malignant exudates frequently contain blood, but this must not be regarded as diagnostic. The only way in which the diagnosis can be established with certainty is by finding small masses of malignant cells preserving their alveolar arrangement intact. The cells themselves are not characteristic, and I must confess myself unable to differentiate between them and some forms of endothelial cells.

7. Examination of cyst-contents.—Cysts are not often tapped now for diagnostic purposes; occasionally, however, this is justifiable, and a knowledge of the nature of the contents of some of the most important may be useful.

(1) **Hydatid cysts.**—Normally the fluid is clear and watery, and contains salts (especially chlorides) but no albumin or sugar. The important elements for diagnosis are the characteristic hooklets; these are occasionally absent, but may usually be found in the centrifugized deposit. Scoleces, too, may be ob-

served occasionally. Sometimes the fluid becomes turbid, or may undergo suppuration. The hooklets, however, are highly resistant, and will usually be found after a careful search.

(2) **Dermoid cysts.**—The content of these cysts is often too thick to pass through a small trocar. It must be remarked that it contains material of a fatty nature, and an examination of the cold material gives a false impression of its consistency during life. Both classes of dermoid cysts, the epidermoid and the teratomatous, contain fatty material, cholesterolin, and desquamated squamous epithelium, which retains Gram's stain. The teratomatous contain in addition hairs, fragments of which may be found in the fluid.

(3) **Ovarian cysts.**—The ordinary cystic adenoma of the uterus contains pseudomucin and paralbumin, substances which are of a thick gelatinous or glairy consistency, and are often called colloid. In addition, there are frequently desquamated epithelial cells (usually columnar), leucocytes, fat, and often some cholesterolin crystals. In the forms of cyst with highly developed papillary systems (cystadenoma serosum) the fluid is thin and watery and contains much albumin. The contents of parovarian cysts vary, being either serous or else glairy from the presence of mucin or paralbumin. When cells occur they are often ciliated epithelium, and this, when present, is very suggestive.

(4) **Pancreatic cysts.**—The examination of fluid from these cysts is often of great importance in diagnosis. The characteristic feature is the presence of trypsin, which may be detected by digesting some of the fluid at 45° C. in contact with shreds of fibrin stained by carmine; if trypsin is present the fibrin will be digested and the dye pass into solution. There are certain precautions necessary in using this test: (a) the incubation should not be at a lower temperature than that indicated, or bacterial action may vitiate the result; (b) trypsin or a similar enzyme is given off from polynuclear leucocytes, and that fact must be borne in mind if many of these cells are present in the fluid under examination.

Enzymes may be absent in an old pancreatic cyst. Altered blood, cholesterolin, fats or fatty acids may be present.

(5) **Urinary cysts.**—When examining cysts from the neighbourhood of the kidney, etc., it is sometimes necessary to know whether they are in communication with the urinary tract. The presence of more than a mere trace of urea

BALANITIS

is the important diagnostic point, and it is advisable that this should be investigated by the Soya bean (urease) method, as substances other than urea give off gas with sodium hypobromite.

W. D'ESTE EMERY.

BALANITIS.—Inflammation of the glans penis and preputial sac.

Etiology.—Balanitis may be venereal in origin, and is then acute, and due to soft sore, hard chancre or gonorrhoea. Non-venereal balanitis, more often subacute or chronic, is due to the decomposition of retained smegma, herpes preputialis, carcinoma of the penis, infection from a leucorrhoea in the female, or the irritation of diabetic urine. A chronic form is also associated with gout. All forms are predisposed to by phimosis.

Symptoms.—The glans is the seat of burning pain and irritation, and can be felt swollen and tender through the prepuce, especially along the corona. If the swollen foreskin can be retracted, a causative lesion is often found. The inner surface of the prepuce and the surface of the glans are red, excoriated and "weeping."

Treatment.—In the *acute* form, if it is possible to retract the prepuce freely, this should be done several times a day and the parts washed with a lotion of potassium permanganate (1 in 5,000). If there is phimosis and retraction is impossible, much the best plan is to slit the foreskin up the dorsum under gas. The cause of the trouble can then be determined, free drainage is allowed, and the condition becomes amenable to treatment. Circumcision should not be done at the same time, but later, should the patient desire it. In the *chronic* form, but not that associated with diabetes, circumcision is indicated after the condition has been cured as far as possible by the application of lead lotion. Circumcision is necessary to prevent further attacks, which predispose to carcinoma of the penis. In diabetes, constitutional treatment is indicated, with the application of zinc ointment to prevent the constant irritation of the part with the urine.

C. A. PANNETT.

BALNEOTHERAPY (see SPA TREATMENT).

BALZ'S DISEASE (see LIPS, AFFECTIONS OF).

BANTI'S DISEASE (see ANÆMIA).

BARLOW'S DISEASE (see SCURVY).

BED-SORES

BASEDOW'S DISEASE (see EXOPHTHALMIC GOITRE).

BATHS (see HYDROTHERAPY; SPA TREATMENT).

BAZIN'S DISEASE (see ERYTHEMA; TUBERCULIDES).

BEDNAR'S APHTHÆ (see STOMATITIS AND GLOSSITIS).

BED-SORES (*syn.* Erythema Paratrimma).—A local destructive and ulcerative process beginning superficially and brought about chiefly by continuous pressure.

Etiology.—Greater care and efficiency in nursing have, happily, made this condition far less common than formerly. The sores appear on points of pressure, such as over the sacrum, trochanters, heels, or ankles. They occur in the bedridden, especially those who are incontinent or who suffer from a nervous disorder in which there is trophic disturbance, such as myelitis. The aged and debilitated are particularly liable to them. The chief cause is persistent pressure against the bed, or a badly applied splint, and their formation is assisted by dirt, or by the skin becoming sodden by sweat, urine, faeces, or other discharges. The excoriation which commonly occurs where two areas of skin lie in apposition, as under the breasts, between the scrotum and the thigh, and between the toes, may develop into a bed-sore.

Symptomatology.—Two varieties are met with—the acute and the chronic. The *acute* form is apt to occur in the subjects of nervous diseases accompanied by trophic disturbance. A red patch forms, and around it the tissues become oedematous and may blister; ulceration follows, or a central dry slough may be produced. The *chronic* sore begins as a dusky purplish patch, which darkens until it is almost black, the surrounding area becoming oedematous and perhaps vesiculated. When the vesicles burst, the dermal papillæ are exposed and are seen to have a greenish hue. A greyish or blackish slough forms and slowly separates. The condition may spread so that large crateriform ulcers are produced, which may extend down to the bone. Occasionally the spinal canal may be opened up and death may follow from septic meningitis. Absorption of poisons from the sore may lead to serious toxæmia, and is a common cause of death in chronic nervous diseases.

Treatment.—Bed-sores may be prevented

by careful nursing. Continuous pressure should be avoided by frequently changing the patient's position. A water-bed should be used for cases such as typhoid fever, where much manipulation is impossible. Parts where pressure is exerted may be protected by air- or water-cushions or rings, or by cotton-wool. The draw-sheet should be kept perfectly smooth and changed immediately it is soiled. The strictest cleanliness must be observed, and special attention should be paid to the washing of the endangered parts with soap and water. They should be dried carefully, dusted with boric or talcum sterile powder, and twice a day gently rubbed with methylated spirit. If a bed-sore appears imminent, it may sometimes be obviated by applying a mixture of brandy and white of egg.

If a bed-sore has formed, pressure must be taken off as described. Its surface must be cleansed by mild antiseptic lotions, and dry absorbent dressings, frequently changed, must be used. Fomentations may be required to encourage the separation of the slough, and lotio rubra or balsam of Peru to stimulate the formation of granulations. Few applications clean the sore so satisfactorily as hydrogen peroxide. Healing may be hastened by allowing a stream of oxygen to play on the raw surface every three or four hours. Garlic either as an ointment (50 per cent. in vaselin) or as a watery solution (1-4) has been advocated.

FREDERICK LANGMEAD.

BELLADONNA POISONING (*see* POISONS AND POISONING).

BELL'S PALSY (*see* FACIAL PALSY).

BERGONIÉ TREATMENT (*see* OBESITY).

BERIBERI.—A disease, or group of diseases, of tropical and subtropical climates, characterized by neuritis of a multiple nature, cardiac and gastric disturbances, and oedema. The condition may occur endemically or epidemically.

Etiology.—The name beriberi, though applied more or less indiscriminately to many of the forms of multiple peripheral neuritis met with in the tropics, probably covers a variety of diseases due to allied though different causes. In this group one may place ship beriberi, beriberi in white troops in India, beriberi in Brazil and South America generally, and the epidemic dropsy of Calcutta. These, though clinically more or less resembling beri-

beri as seen in the East, are not dependent upon rice as their cause, whereas the latter, as shown by Braddon, is. This observer has demonstrated for those parts that it is only people who eat highly polished rice that develop beriberi, while researches have shown that it is not the rice *per se* that produces the disease, but the removal, by the polishing process, of a substance—probably a base of the pyrimidine group—contained in the subpericarpal layers. Funk included this substance among the “vitamins,” and under his classification beriberi, polyneuritis of birds, epidemic dropsy, ship beriberi, scurvy, experimental scurvy and infantile scurvy fall under the heading of “deficiency” diseases, a group to which delayed growth and possibly rickets must now be added. The vitamin or accessory food factor concerned with beriberi is known as “water-soluble B.”

A serious enough deficiency of vitamins, then, in any diet, not necessarily rice, may produce a similar train of symptoms, multiple peripheral neuritis, oedema, etc. As far as the type of beriberi seen in the Malay Peninsula and the East goes, its dependence on the use of highly polished rice seems to be definitely proved. In institutions where the disease has been rife a change of diet from polished to unpolished rice has always quickly stopped the incidence of the infection, and when the latter form of food is used alone beriberi does not appear. Though the classical beriberi, then, is a food-deficiency disease, forms of peripheral neuritis, apart from those due to well-known causes such as alcohol, arsenic, etc., would appear to exist. These are not associated with faulty dietary, and may be infective in origin. Records of such outbreaks have been reported from India, South America, and Mesopotamia (Balfour).

Pathology.—The pathological lesions found in patients who have died of beriberi vary according to whether the form of the disease was “wet” or “dry.” In the former, where there has been much oedema, fluid is found in the pleural sacs, pericardium, and more rarely in the peritoneal cavity. The subcutaneous tissues of the legs and back will also be found to be soft and sodden, due to the infiltrating fluid. The lungs commonly show oedema at their bases and may be congested. As regards the heart, in the acute cases the right side is very much dilated, and both chambers, especially the auricle, are full of dark clotted and semi-fluid blood, this engorgement extending into the large veins of the thorax and neck.

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The left side is sometimes slightly dilated also. In longstanding cases some hypertrophy becomes superadded to the dilatation. The cardiac muscle shows fatty changes and tends to be friable. As regards the other viscera, the liver, spleen, and kidneys may exhibit the usual changes associated with chronic venous congestion, these varying considerably in degree. The mucous membrane of the stomach and duodenum is hyperæmic, and small hæmorrhages are often noted there.

In dry or atrophic cases, the brain and other viscera do not show any macroscopic changes of importance; the wasting of the muscles is apparent; there are no serous effusions; and the heart does not present the extreme engorgement seen in the acute forms. The microscopical changes in the nerves have been worked out by Wright and others. The nucleus of the vagus in the medulla is said to be altered, and the cells of the posterior spinal ganglia and anterior cornua of the cord are also degenerated. The peripheral nerves, though macroscopically normal, microscopically show characteristic degenerative changes. These, briefly, are very similar to those seen in alcoholic and other forms of peripheral neuritis, the vagi and nerves supplying the legs being chiefly affected. All grades may be seen, the medullary sheath and axone in advanced cases completely disappearing. The sensory and vaso-motor nerves are also attacked, and the implication of the latter is said to be chiefly responsible for the œdema, though this must be accentuated by the heart failure.

The muscular fibres, especially in the "dry" atrophic variety, are much atrophied, the striation of the fibres is lost, and ultimately a colloid appearance is seen.

The lesions of beriberi, then, are due to nerve degeneration, the special implication of the cardiac nerves being responsible for the heart symptoms, that of the sciatic and its branches for the paralysis of the legs, and that of the vaso-motor nerves for the œdema.

Symptomatology.—Clinically, beriberi has been divided into "wet beriberi" (beriberi with œdema) "dry beriberi" (beriberi with atrophy and no œdema), and "mixed forms," but as these are only stages or phases of the same disease the more commonly adopted procedure at the present time is to consider the disease under its different types. At the same time, it must be remembered that these gradually merge into each other, and cannot in many instances be sharply differentiated from one

another. Allowing for this, the following classification may be adopted:

1. Larval or rudimentary forms.
2. Ordinary forms—(a) Severe; (b) Mild.
3. Acute pernicious forms.

1. **Larval forms.**—These are by no means uncommon, and may even be missed altogether, as the patient may make little or no complaint. It is quite a usual thing for a batch of cases to be sent into hospital, here in England, from one of the steamers trading with the east, some of those affected being typical cases of beriberi, while others only complain of a little weakness, numbness, or discomfort in the limbs, very often with exaggerated knee-jerks. If these are carefully watched, however, the knee-jerks may be seen to diminish and disappear, and finally definite signs of paralysis may develop. The explanation is simple: the disease is commencing, and irritation of the nerves precedes the degenerative changes found later; in this way the apparent anomaly of the increased reflexes is accounted for. Two things now may happen: either the patient goes on with his diet deficient in vitamins and gets a definite attack, or he is put on a proper food ration and quickly recovers. This can be very easily shown experimentally in the polyneuritis of birds. Pigeons fed on highly polished rice soon develop symptoms, but, if then given vitamins, quickly recover. On the other hand, if these are withheld they progressively get worse and die.

2. **Ordinary forms.** (a) **SEVERE.**—The onset of the disease is generally insidious, premonitory symptoms, consisting of tired feelings, slight breathlessness, some indigestion, numbness in the toes and feet, often having been complained of for some time before the attack becomes fully developed. In other instances the onset of the paralysis is more sudden. The patient has perhaps gone on with his work, feeling ill, but still able to do a little, when one day he finds he is swollen and cannot use his limbs. A casual inspection of such a case shows that the breathing is laboured, œdema is present, perhaps only in the legs and over the sacrum, or, on the other hand, to such an extent that the whole body appears waterlogged with fluid, while attempts to elicit the knee-jerk will generally end in failure. Subjectively, the patient may complain of some difficulty in breathing, discomfort in the epigastrium, cramps in the calves, burning and tingling in the feet and legs, and generally also

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of areas of numbness in different parts of the legs. If the different systems are now examined in detail many other points of interest will be brought out.

Alimentary system.—The tongue does not reveal anything of importance, but a congestion of the fauces has been described. The appetite is usually much impaired, and, if the patient eats much, the epigastric and cardiac distress is quickly increased. Ordinary dyspeptic symptoms, as met with in other gastric catarrhs, may also be encountered. Vomiting, when it occurs, is a sign of very grave significance, as it indicates extensive involvement of the branches of the vagi supplying the stomach. Cases in which this symptom is pronounced invariably end fatally. The bowels are often constipated, though, in other cases, fairly normal stools are passed.

Circulatory system.—Palpitation, præcordial pain, and cardiac dyspnoea are present in varying degree. The apex beat of the heart is diffuse, and percussion quickly demonstrates the great enlargement of the organ to the right. On listening to the sounds, a systolic murmur is usually heard in the mitral area; the second sound is reduplicated at the base, and the intervals between the first and second sounds—the long and short pause—approximate each other in duration, leading to what has been termed an equal spacing of the sounds, a feature very characteristic of the beriberi heart. The action is very rapid also, the rhythm, however, being in the main regular. The special characteristics of the pulse are its rapid rate and low tension. With a heart in such a condition, especially if the patient tries to move or take any unwonted exercise, sudden failure may easily take place.

Respiratory system.—Dullness is found at the bases of the lungs behind when pleural effusion has taken place, and this should be carefully watched in order to prevent it from becoming excessive. Crepitations and rhonchi may be heard over most of the lung areas, increasing as the oedema and congestion become worse.

Urinary system.—Secretion of urine is very much diminished when there is much dropsy, its colour being high; even then, however, albumin is not as a rule present, its occurrence generally meaning coincident disease of the kidneys. When the dropsy begins to pass off under the influence of diuretics and other treatment, the flow rapidly increases and may be excessive for a time. Urea and chlorides are generally stated to be diminished.

Hæmopoietic system.—The blood shows little or no change. The red cells, white cells and hæmoglobin are within or about the normal limits. If anæmia or any marked disturbance in the count is present this will be found to be due to coexisting diseases such as malaria, ankylostomiasis, etc. Differentially, the leucocytic formula is as usual.

Integumentary system.—The marked symptom met with here is the oedema. It commences in the lower extremities, first above the ankles and along the tibia. Thence it generally spreads to the feet, and gradually passes upwards, attacking the body, arms and face. It may be excessive, giving the patient a peculiar bloated or swollen appearance. When in less degree the places where it can best be detected are the tibiae, sacrum and sternum. It is said to be firmer and to pit less readily than that associated with renal or ordinary cardiac disease, nor does it affect the genitals to such an extent. When once it begins to undergo absorption it may disappear with great rapidity; then the skin looks wrinkled and the surface layers commonly desquamate. The atrophy of the muscles may now become apparent for the first time.

Nervous system.—The higher nerve-centres are not attacked. The mental faculties remain active to the end, sight, hearing, taste, and smell are unaffected, and trophic lesions do not occur. The sensory lesions met with in the limbs and elsewhere are anæsthesia, hyperæsthesia, and paræsthesia. Of these, anæsthesia varies greatly in its distribution. It may be limited to the front of the legs, while in other cases areas may be found on the arms, chest and abdomen. A ring round the mouth has been specially noticed by the Japanese. Hyperæsthesia has been noted in the skin, but is uncommon. It is always present, however, in the affected muscles, and may be severe. It is brought out in those situations by applying firm pressure to the muscles implicated—for example, squeezing the calf muscles at once makes the patient cry out. Burning sensations, pins and needles, pricking and formication, form the most usual types of paræsthesia.

Motor functions.—The amount of paresis and paralysis present depends upon the nerves specially affected. The anterior tibial and peroneal nerves are most commonly attacked first, and paralysis appears in the muscles they supply. Cramps may be troublesome before this becomes extreme. Later, as other nerves become implicated in turn, the paralysis

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spreads. After the legs, the arms may become involved and even the muscles of the trunk. The latter event, however, is rare. If the intercostals and diaphragm are paralysed death is apt to take place from failure of respiration. Sudden loss of voice indicates involvement of the laryngeals. The knee-jerks in the early stages of the disease may be exaggerated, later they are completely lost. The superficial reflexes can generally be elicited, though in other instances they also are lost. The muscles quickly waste and, in the atrophic forms, may dwindle to almost nothing. In such cases movement is impossible, the patient becoming completely bedridden. The reaction of degeneration is present. Foot-drop then is often seen. The gait is generally described as characteristic, the toes drag along the ground and the patient, to avoid this, lifts the feet high.

Temperature.—This is not altered. If any rise occurs it is due to complication by malaria or some other disease.

(b) **MILD FORMS.**—In these forms the different symptoms are of a much lighter nature than those described above. Though the cardiac action may be excitable, especially after exercise, the organ is not dilated, and there is no special difficulty in breathing. The œdema also is slight, perhaps only limited to the anterior parts of the tibia, while the paralysis may be insignificant and not sufficient to prevent the patient from using his limbs and walking about. The knee-jerks are generally absent, however, and the muscular power when tested is distinctly diminished.

3. **Acute pernicious form.**—The name “fulminating” has also been applied to this very deadly form of the disease. The patient is seized suddenly with severe dyspnoea, cardiac oppression, tachycardia, and all the signs of a rapidly failing heart. He gasps for breath, is deeply cyanosed, the veins in the neck stand out engorged with blood, and in the space of a few hours he is *in extremis* and quickly dies. The explanation of this form is that the vagi have been specially attacked, their structure being more or less completely destroyed.

Diagnosis.—The affection of the heart, the œdema and the loss of knee-jerks, are the three principal factors by which a diagnosis of beriberi may be made. The other forms of peripheral neuritis have other special symptoms which help to differentiate them—for example, *mercuriorum* of the tongue in the alcoholic, diarrhoea and *insanabile* pigmentation in the arsenical, the blue line

on the gums and colic in the lead, and the throat in the diphtheritic. In addition to these, however, diseases in which œdema is met with must be distinguished. *Bright's disease* will be accompanied by albumin and casts in the urine, but no neuritis, while *heart disease* will exhibit the characteristic murmurs, also in the absence of neuritis. Cases with signs of neuritis, absent knee-jerks and pain on grasping the calves, may, if albumin is also present, furnish difficulties. Further, it is quite possible for cardiac and renal cases to develop beriberi, when a correct diagnosis is not so easy. On the whole, however, if the case is investigated thoroughly, no great difficulty arises in saying whether it is one of beriberi or not.

Prognosis.—This largely depends on whether the patient can be taken into hospital. If he is allowed to go about with little or no attention, death is very liable to occur. Cases admitted into the Albert Dock Hospital, in London, if they survive the first three days, almost invariably recover. Acute fulminating cases die, whatever is done, and so also do cases where vomiting is a prominent symptom. Loss of voice, indicating paralysis of the laryngeal muscles, is a grave sign.

Treatment.—Prophylactically, highly polished rice should not be allowed as the sole diet. In theory such a procedure seems simple, but in practice it is not so. Many natives do not like unpolished rice, and refuse to eat it. Future generations will gradually, no doubt, become educated to this point, but, for the present, it may be necessary to add substances rich in vitamins to the polished rice or other defective rations. To this end Cooper advises egg-yolk, heart-muscle, liver, nuts, barley, and lentils; heart and liver, according to him, containing the antineuritic substance in high concentration. Extracts of rice polishings, yeast 2 oz. daily, beans (*katjangidjo*), and “Marmite” in one-cube doses twice a day as an alternative to dry yeast, have also been recommended.

When the disease occurs it is essential that the patient be confined strictly to bed and, if at all bad, he should be prevented from sitting up or moving suddenly. This complete rest in bed, with the change of diet, is often all that is required. Where dyspnoea and cardiac distress are prominent, and the right side of the heart is much engorged, bleeding may be tried. Amyl nitrite will also help to relieve the oppression, and, after the urgent symptom have subsided, digitalis in small doses may be

BIER'S TREATMENT

administered. A dry diet is essential for cases with oedema, and to this should be added rice-polishings, yeast, or katjangidjo beans. Afterwards, when the acute stage is passed, the paralysis and atrophy of the muscles must be attended to, massage and electricity being the best means for this purpose. Once the heart has become normal the patient may be allowed up, but care must still be exercised till all signs of paralysis have disappeared. The patient finally should be warned against going back to his old diet, as otherwise he may get another attack.

G. C. Low.

BIER'S TREATMENT (Artificial Passive Hyperæmia).—A method of producing passive congestion by mechanical means, and thereby imitating the hyperæmia and serous exudation resulting from inflammation. It is especially applicable to diseases of the limbs, but may also be used for sinuses, carbuncles, and other inflammatory lesions elsewhere. In the treatment of cellulitis, arthritis—whether suppurative, rheumatoid, or tuberculous—whitlows, and other affections of the limbs, a broad, soft rubber bandage is bound round the limb on the side proximal to the lesion, sufficiently tightly to cause reddening and swelling below, but loosely enough to prevent cyanosis, pain, or interference with the pulse in the artery. It is usual to apply the bandage at first for a short time (half to one hour) until the degree of discomfort and the effect are observed, but with proper precautions it may be allowed to remain for the greater part of the twenty-four hours. Renewal of the treatment at short intervals is more satisfactory than a continuous long application. For the treatment of local inflammatory conditions of the trunk or head and neck special suction cups have been devised. The method is similar in principle to that by Wright's salt solution, with which it may be combined. **FREDERICK LANGMEAD.**

BILE-DUCTS, CONGENITAL STENOSIS OF.—A condition characterized by partial or complete occlusion of the bile-ducts, accompanied by cirrhosis of the liver.

Etiology.—The causation is obscure. One view is that there is a failure on the part of the ducts to form a proper lumen, and that this is followed by cholangitis, which leads to their complete obliteration, with subsequent cirrhosis of the liver. The other view is that there is primary cirrhosis of the liver caused by some toxin derived from the mother, and

BILE-DUCTS, STENOSIS OF

that obliterative cholangitis is secondary. Against the first view are the facts that the stenosis varies much in position, and that other developmental errors are seldom present. Cirrhosis, too, is a rare sequel to biliary obstruction and, when it does follow, it is multilobular and not unilobular, as in this disease. In favour of the second view is the fact that cirrhosis has been met with occasionally without any stenosis of the bile-ducts. In some instances several children in the same family have been affected, and there seems to be a very close relationship to grave familial jaundice—a form of toxæmic jaundice, in which the less acute cases may show early cirrhotic changes in the liver. In both diseases the parents usually appear to be healthy.

It is probable that in affected infants there is some peculiar susceptibility of the liver to toxins derived from the maternal blood, which would account for the familial tendency and for a case in which twins were born, one of whom died of the disease while the other remained healthy.

Whatever be the cause, it is not syphilis, though there have been one or two cases of syphilitic obstruction to the bile-ducts recorded in infants.

Pathology.—The site of the obliteration varies considerably: sometimes it is near the ampulla of Vater, at others it is in the intra-hepatic part of the ducts. Partial or complete occlusion may occur at several different points. In some cases portions of a duct completely disappear. Round the stenosed areas there is increase in fibrous tissue, and the lumen of the duct shows inflammatory changes. There may be inflammatory thickening of the wall and mucous membrane of the gall-bladder. The liver is large and dark-green in colour, and its surface is leathery. It is very firm on section. Microscopically the cirrhosis is wholly or chiefly unilobular. The majority of the liver cells are normal, but some are necrosed.

Symptomatology.—The child usually appears to be healthy at birth. The first and most important sign of the disease is jaundice. This may be present at birth, but more often appears between the second and fourteenth days. It gradually deepens and becomes intense, though it seldom assumes the deep-green colour seen in prolonged jaundice in adults. At first there may be fluctuations in its intensity. To begin with, normal meconium is passed, but the stools soon become white and are often hard and dry. The urine contains

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bile pigment and stains the napkins. The liver and spleen grow large and hard. At first the child remains well nourished, but, if it survive many weeks, becomes much wasted. Hæmorrhages are common. They occur most often from the umbilicus, but epistaxis, hæmatemesis, bleeding from the bowel, and purpura are not infrequent. Early hæmorrhage usually means early death. Death takes place between the seventh day and the eleventh month, generally in the third or fourth months, and is usually due to toxæmia marked by increased drowsiness, coma and convulsions, or to some intercurrent disease.

Diagnosis.—At first the diagnosis is difficult. The onset is indistinguishable from that of *icterus neonatorum*, but the increasing jaundice and loss of colour in the stools soon show the presence of a more serious condition. The occurrence of hæmorrhages distinguishes it from catarrha' jaundice, which is rare at this age. Grave familial jaundice has the same date of onset, but is very rare in England. In this disease the stools generally remain coloured, severe anæmia may be present, and its course is more rapid. *Jaundice due to infection from the umbilicus* causes more severe constitutional symptoms accompanied by fever, and the septic focus is generally recognizable. The presence of vomiting and diarrhœa also serves to differentiate it. *Winckel's disease*, due to intestinal infection, also follows a more severe and rapid course, and is characterized by hæmoglobinuria, cyanosis, and diarrhœa. *Hæmolytic jaundice*, which is often familial, seldom shows itself so early. The jaundice is lighter, the faces are dark, the urine, though highly coloured, contains urobilin but no bile pigment, and the red blood-cells are fragile.

If the child lives for some weeks, the enlarged hard liver and spleen distinguish congenital stenosis from all these conditions except hæmolytic jaundice.

Prognosis.—In cases proved to belong to this group, death invariably takes place. Familial cases and those with early hæmorrhages are usually fatal within a fortnight of birth. In milder cases the infant may survive for two or three months, but never as long as twelve.

A few cases of prolonged jaundice have been met with in families in which other children had this disease and have recovered. It is possible that they may have been slight cases of the same disease, with cirrhosis of the liver only.

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Treatment.—Operation is not justifiable. The stricture can be relieved in very few cases, and there is great risk of death from hæmorrhage, and even if the stricture is relieved the cirrhosis causes a fatal issue. No medical treatment can cure this disease, but it is wise in any given instance to try the effect of mercurial treatment in the hope that it may be one of the rare cases of syphilitic obstruction. Life may be prolonged slightly by subcutaneous infusion of saline or by the administration of sodium bicarbonate or citrate in large doses.

E. A. COCKAYNE.

BILE-DUCTS, INFLAMMATION OF
(see CHOLECYSTITIS AND CHOLANGITIS).

BILE-DUCTS, OBSTRUCTION OF (see JAUNDICE).

BILHARZIASIS (see SCHISTOSOMIASIS).

BILIARY CALCULI (see GALL-STONES).

BILIARY COLIC (see GALL-STONES).

BILIOUS ATTACKS. The term "bilious attack" has a popular rather than a scientific meaning. It denotes, however, a fairly definite group of symptoms. The patient is dull, languid, irritable, and often depressed. There is a great distaste for food, usually accompanied by thirst. Nausea and headache are present, and, in severer cases, vomiting. At first food is returned; later the vomitus may consist chiefly of bile and mucus. Sometimes the vomiting rapidly relieves the symptoms, but this is by no means always so. The tongue is coated and the breath foul. Constipation is the rule, but it may be replaced or succeeded by diarrhœa. An icteric tinge may be noticed in the conjunctivæ. The urine is scanty, high-coloured, and laden with urates. The condition is common in children and in gouty people. Frequently it can be ascribed to a definite indiscretion in diet, such as the taking of an indigestible meal, or one which is too large or too rich, or an alcoholic excess may be the cause. In these circumstances the term "bilious attack" is practically synonymous with "acute dyspepsia." Some patients exhibit an idiosyncrasy for certain kinds of food and develop an attack if they transgress rules of diet which are specially applicable to themselves. In other instances the effect seems to be a cumulative one; no indiscretion can be remembered, yet the elimination of a particular form of food from the dietary may

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prevent recurrence of the attacks or diminish their frequency.

Attacks may also be due to uncorrected errors of accommodation or refraction. Slight and unsuspected astigmatism is said to be a common cause.

Psychical disturbances play a part in certain cases. There are some persons for whom any exceptional excitement leaves an attack in its wake. Between bilious attacks and migraine there is a close relationship. Not only do the symptoms of a bilious attack form part of the clinical picture of migraine, but such attacks in childhood may be replaced by genuine migraine at puberty. Cyclical vomiting is so nearly allied that it is impossible to differentiate between them in many cases. In cyclical vomiting, however, the attacks bear no relation to diet, and the degree of toxæmia may be much more profound (*see VOMITING, CYCLICAL*).

It is important to bear in mind that chronic and subacute appendicitis may manifest themselves clinically as "bilious attacks."

Treatment.—No hard-and-fast rule can be laid down by which the attacks can be prevented. An endeavour must be made in each case to ascertain in what direction the fault lies. The diet and habits of the patient must be regulated, a free action of the bowels ensured daily, and any defect of refraction or accommodation corrected. Regular administration of alkalis after meals will often lessen the frequency and severity of the attacks. In gouty subjects an after-dinner blue pill, followed by a saline in the morning, is often a useful preventive measure. For the attack itself the patient should remain in bed. A period of starvation is better than any food. The most efficacious remedy is calomel or some other mercurial purge followed by salines. When the appetite returns, food should be given sparingly at first. Solids are usually borne better than milk. Fats, except in the form of butter, should be avoided until convalescence is well established.

FREDERICK LANGMEAD.

B. I. P. P. TREATMENT (*see WOUNDS, TREATMENT OF*).

BIRTH INJURIES.—Birth injuries may occur as the result of normal labour, but are much more frequently due to manipulative interference. Prophylaxis lies to a large extent in careful antenatal examination, with a

view to ensuring that the infant does not grow too large for the pelvis in question, and to converting, where possible, abnormal into normal presentations. If labour is in progress, preventive treatment consists in allowing and encouraging the natural processes, or, if instrumental or other artificial means of delivery must be undertaken, in performing these without undue haste or force, and changing, for example, posterior positions to anterior, manually, before making traction with forceps.

The commoner birth injuries are the following:—

1. **Caput succedaneum.**—This is a diffuse swelling of the scalp, produced over the part of the fetal head that presents during labour. It is considerable when there has been difficulty in the passage of the head through the pelvis. It is an œdema of a part free from pressure produced because the ring of tissue above it is so firmly compressed as to retard the return flow of blood from the part. By its position the presentation of the child can be confirmed after birth. The swelling is present at birth, and gradually diminishes in size, disappearing in twelve to twenty-four hours. No treatment is required.

2. **Cephal hæmatoma** is sometimes confused with caput succedaneum. A defined fluctuant swelling on the vault of the cranium caused by hæmorrhage under the periosteum, it may be single or multiple. It is limited by the sutures. Its greatest size is reached within two to three days after delivery. Absorption is slow, taking sometimes as long as three months. During absorption a hard ridge forms round the circumference of the swelling, and may give a false impression of a depressed fracture. No treatment is called for.

3. **Depression of the bones of the skull or fractures** may occur, the latter almost always due to instrumental delivery and associated with hæmorrhage, extra- and intracranial. Depressions may be reduced spontaneously or may be aided by gentle pressure of the skull in their long axis. If they remain, operative measures for the elevation of the bone should be undertaken, as in all cases of depressed fracture.

4. **Intracranial hæmorrhage** may occur apart from fracture, and sometimes in spontaneous deliveries. The clot formed is almost always subdural. The symptoms take from three to four days to develop. The prognosis is bad, but not absolutely so. Help may be given by the use of an ice-cap, sedatives per rectum,

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and the injection of human or horse-serum. Syphilitic infants are subject to such hæmorrhage. In infants who survive there is great probability of an abiding spastic paralysis.

5. **Facial paralysis** is usually of a temporary nature, passing off in a few days; it is due to pressure of the anterior blade of the forceps upon the 7th nerve, as it emerges from the stylo-mastoid foramen.

6. **Hæmatoma of the sterno-mastoid muscle.**—A healthy muscle is not easily injured. Rupture of certain fibres may occur as the result of traction associated with torsion of the head, or the muscle may be crushed with forceps, and hæmorrhage occur into its substance. The head is turned towards the affected side. Spontaneous recovery is the rule, and, unless infection takes place, there is no shortening.

7. **Injuries to nerves causing paralysis.**—The most common injury is that which produces "Erb-Duchenne paralysis." The 5th and 6th roots of the brachial plexus are dragged upon, or pressed with the forceps, and, as a result, the arm lies limp at the side with the hand rotated inwards. This subject is dealt with more fully under SPINAL NERVES, LESIONS OF.

8. **Fractures of the clavicle, humerus, femur, and other bones.**—Fracture of the clavicle in its outer third is not uncommon. A small pad of cotton-wool should be placed in the axilla, and the arm bandaged to the side. The humerus is sometimes broken in delivery of the arm in breech cases; separation of an epiphysis readily occurs. The femur is also sometimes broken in breech delivery, but this should not occur; here again separation of an epiphysis is the more common injury. The best method of treatment is to bind the thigh against the abdomen, using a light splint on the outer side of the thigh.

FRANCES M. HUXLEY.

BIRTH PALSY (see SPINAL NERVES, LESIONS OF).

BISMUTH MEAL (see X-RAYS, DIAGNOSTIC USES OF).

BLACKWATER FEVER (*syn.* Hæmoglobinuric Fever).—This dreaded disease is characterized by the urine containing much hæmoglobin and methæmoglobin in solution. Best known in Tropical Africa, it occurs also in South America, in Mesopotamia, in the Balkans, in some of the Mediterranean islands, and in parts of India, the Malay States, and Solomon

Islands. There is reason to believe that it is more common now than formerly, though statements that it did not occur in certain countries, such as India, must be received with caution, as often this statement should be modified to "was not generally recognized."

Etiology.—The causation of the disease is not known. Special parasites have been described, but none is generally admitted to be the cause. There is much blood destruction, and portions of cells may be found included in phagocytes; many believe that some of the bodies described as parasites are such included remnants of the red cells.

The disease occurs in persons who have previously had malaria, and malaria parasites may be found in the blood if it is examined before the onset. They may appear again some days after recovery, but are rarely seen during the attack. Those found after the attack are usually benign tertian. An increase in the proportion of large mononuclear leucocytes, and occasionally leucocytes containing pigment, are met with during and after an attack; in fatal cases recent pigment may be found in the connective-tissue cells in the liver and other organs. There is clear evidence that in some cases quinine may, after malaria, produce hæmoglobinuria; but blackwater fever is not a direct poisoning by quinine, nor is it closely dependent on the dosage of that drug.

The conditions inducing blackwater fever appear to be (1) regular use of quinine, (2) an infection with malaria, severe or slight, and of any species, (3) an irregular course of quinine, usually with (4) chill or exposure.

It cannot be said that these conditions are invariable, but they are usual. Cases may occur, it is stated, in which no quinine has been taken, and rarely but more commonly in persons who are regularly taking quinine without any increase or alteration of the dose. These cases are exceptional. A slight alteration in the dose sometimes suffices. A man who had been taking 5 gr. a day regularly for eighteen months changed his method to 10 gr. twice a week, and after the change developed his first attack of blackwater fever. In another case a course of intramuscular injections of 9 gr. of quinine twice a day was changed to 10 gr. three times a day by the mouth, with the same sequel.

Most of the cases met with in England are singularly like each other as regards history. Quinine in 5-gr. doses has been taken regularly in West or East Africa, Burma, or the Malay

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States. On the way home quinine is discontinued, with the result that a mild attack of malaria follows. The use of quinine is now resumed, but only when the patient feels there is need for it, or when fever is coming on, and is discontinued when the fever is under control, again to be taken when he feels feverish. One day or night he gets thoroughly chilled, whether shooting, fishing, dancing, or going out, and takes perhaps a 10-gr. dose of quinine; a few hours later he is down with blackwater fever. These attacks in England are sometimes the first, but they need not be so, and in persons who have had blackwater fever, and subsequently malaria, special caution as regards the use of quinine has to be exercised on account of their special liability to the disease.

It is not to be disputed that there are persons who, after one attack of blackwater fever, may have others induced by small or large doses of quinine, but in the great majority of cases the relationship of malaria, quinine, and blackwater fever is a complex one.

The greater prevalence of blackwater fever, during recent years, in places where it was previously rare or unknown, is singularly associated with the increased frequency of a regular use of quinine as a prophylactic measure; though in individual cases the actual attack occurs after the cessation of the regular use of quinine.

Symptomatology and pathology.—The onset is sudden, usually with a rigor, and often with pain in the liver or the renal region. This pain may be severe and persistent, but more often lasts only an hour or two. Shortly afterwards there is an urgent call to micturate and the urine appears black. When diluted, or when the urine is shaken up in a bottle, its froth is pink from the dissolved hæmoglobin. Examined by the spectroscope after dilution, the bands of oxyhæmoglobin and sometimes of methæmoglobin are found. If the urine is allowed to stand, a large flocculent deposit appears, and the superjacent fluid is clear in thin layers, and when diluted, in thicker. The deposit is mainly amorphous, but numerous casts coloured with hæmoglobin are found and, exceptionally, red blood-cells. Later on in the disease there are epithelial cells from the bladder, the ureters, and the pelves of the kidneys. After a time the urine clears, but for a variable period of days contains albumin and for weeks, or even longer, casts of the renal tubules.

The course of the disease varies greatly.

Sometimes the urine clears in a few hours; one, two, or three days elapse in severe cases, and sometimes, in fatal ones, the urine does not clear for over three days.

Another form consists of a series of mild attacks. The urine clears or nearly clears in one or two days, but after a brief interval again becomes loaded with hæmoglobin, and again clears. In these relapsing cases the urine may not be quite clear for a week or more.

The hæmoglobin is derived from breaking-down red corpuscles, and free hæmoglobin is found in the blood-serum. In the liver and renal cells hæmoglobin granules are present; these cells contain iron in a state of loose combination, so that the reactions for inorganic iron are obtainable. All the tissues are stained a yellowish colour from the decomposed hæmoglobin in the blood-serum, and this colour is visible in the conjunctiva and skin. In other words, a hæmatogenous jaundice is present.

The rapid blood destruction soon leads to profound anæmia, so that in a three-day attack the red cells may be little more than one million per c.mm., instead of about five millions.

Prominent symptoms, in addition to the "blackwater," are therefore anæmia and jaundice. The dissolved hæmoglobin appears to exert a toxic effect, for pyrexia, frequent rigors, and sometimes delirium are concomitants while this blood destruction is taking place. Towards the end of the period and after it, the sighing, breathlessness, and restlessness of the profound anæmia are to be noted.

The chief danger, however, is not the anæmia and jaundice, but is due to the mechanical obstruction of the renal tubules attributable to the deposition in them of the albuminoid deposit from the urine. This may take place along the whole length of the tubules, but more frequently the bulk of the deposit accumulates in the pyramids. Obstruction of a large number of systems of tubules may then occur, and more or less complete suppression of urine result. The suppression may supervene early, but usually occurs when the urine becomes less loaded with hæmoglobin and begins to clear, for at that time the natural flow of urine diminishes. The hæmoglobin dissolved in the blood acts as a powerful diuretic, so that, at first, two, three, or even more times the normal secretion takes place. As the overstimulated renal cells cease to act and the amount of fluid withdrawn from the body

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decreases, there is danger of a diminution of the flow, and therefore of such deposits taking place and blocking the tubules.

When suppression sets in, the prognosis becomes much more unfavourable. The suppression is rarely complete, and, if a catheter be passed, a few drachms of urine may be obtained in the twenty-four hours; but it may be several days before there is enough urine in the bladder to be passed voluntarily. Death usually occurs within two or three days of the onset of suppression, but in rare cases may be deferred for a week or so. The casts blocking the tubules being soft, there is a possibility that they may become dislodged and the flow of urine re-established, for the renal structure is quite healthy. This re-establishment of the secretion is not rare after eighteen or twenty-four hours of suppression, and may occur with recovery of the patient after five days of suppression, or, as in one remarkable case, after eight days. This complication is the main cause of death in blackwater fever. Anæmia, particularly if no special precautions are taken and the patient is allowed to sit up and move about, is perhaps the second most common cause, usually by sudden cardiac failure. The severe and continuing fever and hyperpyrexia, which may persist for many days after the urine has cleared, may prove fatal. Inter-current disease, such as pneumonia, hepatitis, and liver abscess, are all occasional sequelæ that may have a fatal termination.

Treatment.—As no organism is known, there is no specific treatment, though it is claimed by some that salvarsan and its modifications give good results. With a proper understanding of the pathology of the disease, however, the main dangers can be avoided. The disease tends to run a limited course, and causes no organic lesions, so that recovery is rapid.

We cannot check or control the hæmolysis, but we can prevent the deposit in the renal tubules of the material which forms the casts. It is essential to flush the renal tubules by maintaining a free flow of urine from the onset of the disease. The hæmoglobin dissolved in the serum acts as a powerful diuretic, but enough fluid must be supplied to cause the flow of urine to be at least two or three times the normal amount, especially in the earliest stages of the disease, when probably the block commences. As the normal rate of secretion averages about 2 oz. per hour, and as the rigors, fever, and sweating dispose of some fluid, the

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aim should be to supply 8–10 oz. of fluid per hour from the onset of the disease. It can be given by the mouth in small quantities—say one wineglassful at a time, slowly sipped. If vomiting precludes fluid from being taken by the mouth, it should be given by the rectum. Eight ounces with a little salt (a teaspoonful to a pint of water) should be given warm and injected very slowly and as high up as possible. If 8 oz. fail to be retained, more success will probably attend the injection of 6 oz. The injection should be repeated hourly if no fluid is taken by the mouth; but even if fluid can be given orally, an injection every two, four, or six hours will be beneficial. Some authorities prefer that no fluid should be given by the mouth, but all by the rectum.

If commenced early, this line of treatment usually prevents suppression. If not, it should still be adhered to, and large subcutaneous injections of normal saline administered in addition. During this stage no aperients should be given, as it is desirable that all fluid should pass through the kidneys.

The danger of death from anæmia and cardiac failure is much diminished by careful nursing and absolute rest, both during the late stages of the disease and for some days after the urine has cleared.

Among drugs, opium is of high value, particularly when suppression has been allowed to supervene. Morphine either by the mouth or hypodermically ($\frac{1}{2}$, $\frac{1}{4}$, or $\frac{1}{8}$ gr.) will check the vomiting and allay the restlessness; it can be continued safely in small doses for several days; during the sleep that follows, the flow of urine will sometimes be re-established. Calomel is used by some. After the urine has cleared there is often some secondary fever, which is best treated by purgatives. The blood must be examined, however, for the fever may be malarial and require small doses of quinine commencing with $\frac{1}{2}$ gr. and slowly increased.

C. W. DANIELS.

BLADDER, EXAMINATION OF (see CYSTOSCOPY).

BLADDER, INFLAMMATION OF (see CYSTITIS).

BLADDER, NEW GROWTHS OF.—By far the most important tumours of the bladder are the villous papilloma and the malignant epithelial growths, which together constitute over 90 per cent. of all vesical tumours. The other epithelial growths (ade-

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noma and cholesteatoma) and all the connective-tissue tumours (fibroma, myoma, angioma, and the various types of sarcoma) are so rare that they need no further mention.

Etiology.—Very little is known on this point. It has been suggested that as bladder tumours are usually situated near the ureteric orifices, they may be due to some irritating constituent of the urine excreted by the corresponding kidney; but against this is the fact that the urinary stream is directed away from, and not towards, their usual site. Workers in aniline dyes, and those affected with schistosomiasis, are especially prone to suffer from them. This applies both to papilloma and to carcinoma. The age-incidence is usually from 30 to 50 for papilloma, and from 40 to 60 for carcinoma. Papillomata are occasionally met with between the ages of 20 and 30; before this they are very rare. Nearly 80 per cent. of all bladder tumours occur in males.

VILLOUS PAPILLOMATA OF THE BLADDER

These are usually cited as being typically benign growths. That description is true from a histological point of view, but clinically they exhibit many features which suggest either local malignancy or that they constitute a precancerous condition.

Pathology.—They may be pedunculated or sessile: generally the stalk is so short that the lower branches are in contact with the bladder wall; they are then termed sub sessile. From the central stalk numerous branches are given out in all directions; these divide and redivide until the terminal twigs are long, thin filamentous processes. Each process is composed of a central core of connective tissue, containing one or more large blood-vessels, and covered by transitional epithelium. The growths are of a whitish-grey colour, and may be of any size up to that of a Tangerine orange. If the villi are long the tumour has the appearance of delicate moss or seaweed; if they are short and close-set it resembles a cauliflower. The shorter they are the more likely is the growth to be malignant. If the epithelial covering is composed of many layers of cells, which are irregular in shape and size, and which do not follow the typical arrangement of transitional epithelium, the tumour should be considered malignant, even though no definite down-growth of the epithelial cells through the basement membrane can be found. Such tumours show a decided tendency to become multiple in the early stages of the disease, and always

infiltrate the bladder-wall in the later, when they are typical malignant epitheliomata.

The most common site is above and to the outer side of the ureter. Very often the orifice is hidden by an overhanging growth. The tumours are occasionally multiple when the patient is first seen, but as their cells have the power of grafting themselves on normal mucous membrane, and so producing "contact growths," it is probable that they all originate from a single tumour; in these cases the largest growth is usually situated near the ureter. They tend at first to surround the trigone, involving the lower part of the lateral and posterior walls of the bladder, but ultimately they spread over the entire mucous membrane. The trigone itself and the apex of the bladder are the last parts to be involved.

Symptoms and diagnosis.—Hæmaturia is usually the only symptom. The patient, for no reason that he can assign, passes blood-stained urine. Generally the hæmorrhage is fairly copious. The first portion of urine passed may be nearly clear, but it becomes more and more bloodstained as the bladder is emptied. Short irregularly-shaped clots may be passed. There is no pain, except when clots are passing down the urethra. After a period of a few hours to a couple of days the bleeding ceases, only to return later. At first the intervals between the attacks of hæmorrhage are to be reckoned by weeks or even months, but they tend to become progressively shorter until some blood is passed every day. The patient becomes anæmic, but not emaciated. Occasionally fragments of growth are passed, or may be snared in the eye of a catheter. It is unwise to trust to the microscopical examination of these fragments, as by this means cancer cannot be excluded. Sometimes there is difficulty in micturition, or even retention; this may be due to bloodclots in the bladder, or to a growth blocking the internal meatus. A tumour situated at one ureteric orifice may cause a hydronephrosis from back-pressure. The symptoms of a swelling in the loin, and hæmaturia, may then lead to the mistaken diagnosis of a renal neoplasm. Cystitis seldom arises spontaneously, but these cases are exceedingly liable to become infected by instruments. The diagnosis is completed by means of the cystoscope, which enables the size, position, number, and nature of the growths to be determined. If cystoscopy fails, as it may do when the tumour is so large that it almost fills the bladder, an exploratory cystotomy should

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be performed. Occasionally a large papilloma can be felt by combined rectal and abdominal palpation.

Treatment consists in operation or in destruction of the tumours by fulguration (diathermy, q.v.).

Results.—Until recently the open operation has been followed by a large number of recurrences (28 per cent., Rafin), but most of these recurrences are undoubtedly due to implantation of tumour cells at the time of the operation. The recurrences are frequently multiple, sessile, and show a great tendency towards malignancy, so that the patient's condition may be made worse by the operation. Since this has been recognized, the percentage of recurrences has fallen enormously. My own figures are as follows: Excluding all cases operated on within the last three years, I have had 3.3 per cent. of recurrences after excision of single papillomata, but in every case in which two or more growths were present there has been a rapid recurrence. However, these were with one exception recurrent tumours when I first saw them. I have had 12 per cent. of recurrences after diathermy (again excluding cases treated within the last three years), but with two exceptions all these were easily cured by the same means. One was a case of extensive recurrence after four previous operations, and was really malignant; in the other a prostatectomy was performed and the growth removed at the same time. I now hold that diathermy is the best treatment for all benign papillomata, but the patient should return for cystoscopy every six months for three years. If a small recurrence is noticed it can be destroyed immediately.

MALIGNANT TUMOURS OF THE BLADDER

These tumours are best divided clinically into villous and infiltrating growths. The distinction is not absolute, as in the late stages of the disease all the villous malignant tumours infiltrate the bladder-wall.

Pathology.—(1) **Villous growths** may be pedunculated or sessile. (a) Malignant *pedunculated* papilloma may arise from a benign papilloma or may be malignant *ab initio*. In the latter case the villi are stunted and closely packed, giving the tumour the appearance of a cauliflower; occasionally they are so small that they are not recognizable to the naked eye, the growth then appearing solid and resembling a mulberry. Microscopically the villi are covered by many layers of epithelial

cells, which are extremely irregular in shape and size, and do not follow the regular arrangement of transitional epithelium. Masses of epithelial cells may be found in the lymph-spaces of the stalk, which is then thick and hard, but on the other hand the pedicle may not be infiltrated, and the diagnosis cannot be made from its condition alone. In many cases these malignant papillomata resemble the benign growths very closely, and the diagnosis can only be made by means of the microscope.

Malignant disease developing from a benign papilloma may only be recognized under the microscope, when cancerous changes are observed in certain villi, or the transition may be obvious to the naked eye. In the latter case one portion of the growth appears to be typically benign, whilst another is nodular and infiltrating. On account of this, the discovery of apparently benign villi in the urine does not preclude malignancy. The earliest malignant changes are found in the villi themselves, and not in the stalk, as was formerly supposed.

(b) There are two varieties of malignant *sessile* growths. The first is a luxuriant growth, with long villi, that spreads over large areas of the bladder-wall; it is very friable, gives rise to severe hæmorrhage, and, if the bladder is infected, sloughs extensively. At first there is little infiltration of the bladder-wall. The second type is the "nodular tumour." To the naked eye this growth does not appear to be villous. The processes are short, and closely packed, and have a tendency to anastomose together, and the true nature of the tumour is only revealed by the microscope. It has a sharply defined edge, with a rolled margin, and a somewhat depressed centre. Often cracks or fissures run across it, and from these the bleeding arises. The tumour grows slowly, but the whole thickness of the bladder-wall under it is infiltrated early. A bloodclot, slough, or cap of phosphates is often found adhering to the centre of the tumour.

(2) **Infiltrating growths** are characterized by a widespread infiltration of the bladder-wall without much projection of the tumour into its cavity. They are squamous-celled, cylindrical-celled, and alveolar carcinoma. The tumours are slightly raised, irregular and nodular. Often the mucous membrane is puckered and thrown into irregular folds. At first it is intact; later on it becomes ulcerated, and only then does the hæmaturia begin.

Metastases.—The glands most frequently involved are the internal iliac, and after these

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the lumbar. Metastases at a distance are not common, but have been found in the liver, lungs, and spleen, and in bones, especially the vertebrae.

Symptomatology.—The initial symptom is usually hæmaturia, which occurs in attacks just as in benign papillomata. Soon cystitis supervenes, generally spontaneously. Once this has set in, the clinical picture is completely altered. The symptoms then are frequency of micturition, pain, and hæmaturia. The frequency is both diurnal and nocturnal. The capacity of the bladder is limited, and when this limit is reached an uncontrollable desire to micturate is experienced. This symptom readily increases in severity, so that a patient who at first can hold his urine for three hours soon finds he must pass it at two-hourly, hourly, or even half-hourly intervals. At the same time the call to micturate is imperative, and if it is not responded to immediately the urine will be passed involuntarily. The pain comes on as a sudden desire to pass water, and continues throughout the act as a severe scalding along the urethra, which lasts for several minutes after the bladder has been emptied. Often, especially if the tumour is covered with a cap of phosphates, pain at the tip of the penis immediately after micturition is a marked symptom. Shooting pains down the thighs due to involvement of the sacral nerves, or a lumbar ache from involvement of the ureter, may be present. With the onset of cystitis the type of hæmaturia also changes. Instead of being intermittent it becomes almost continuous. Some blood is passed in every act of micturition, though the quantity varies greatly. Straining or tenesmus usually results in the passage of almost pure blood. In the later stages of the disease the urine is characteristic; it is of a deep-brown colour from altered blood, very foul, and when it has stood for some time a copious sediment of pus, shreds of necrotic tissue, phosphatic debris, and small bloodclots is deposited.

Diagnosis.—In the earlier stages of the disease the diagnosis is made by means of the cystoscope. This instrument should be used as soon as possible in every case of hæmaturia, as in this way alone can malignant tumours of the bladder be diagnosed when they are operable. Later on the bladder base will be found to be indurated on rectal examination, or a swelling palpated above the pubes. No cancer of the bladder is palpable when it is in the operable stage.

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Course.—The duration of the disease varies with the type of tumour. In the infiltrating growths death takes place within eighteen months or two years, in the nodular type the patient may live a year or so longer, whilst if a benign papilloma becomes malignant the duration of the disease may be many years. Death is due to exhaustion from pain and loss of sleep, to septic pyelonephritis, or to septicæmia.

Treatment.—(1) **Radical treatment** consists in partial or total cystectomy. The mortality after partial cystectomy for small circumscribed growths is about 10 per cent., and the recurrences after it about 50 per cent. The mortality after total cystectomy is almost 50 per cent. Fulguration gives good results in certain cases of malignant papilloma, in which the stalk and base are not infiltrated, but does more harm than good in all other cases.

(2) **Palliative.**—About two-thirds of the cases are inoperable when first seen. Radium is often of value, but it should be buried in the growth; radiation through the skin has little value. In extreme cases of pain and frequency of micturition the ureters may be brought out on to the skin, and the urine so deviated from the bladder. For severe hæmorrhage a suprapubic cystotomy may be necessary. Morphia, diuretics, and bland fluids should be given freely. Little can be done by washing out the bladder with antiseptic lotions, as it is absolutely intolerant, and this treatment only increases the patient's distress.

J. SWIFT JOLY.

BLADDER, RUPTURE OF (see ABDOMINAL INJURIES).

BLADDER, TUBERCULOSIS OF.—As a primary focus of tuberculous infection in the body, vesical tuberculosis does not exist, but it is said to be primary or secondary according to whether it occurs as the original focus in the genito-urinary system, or secondary to a tuberculous focus in the kidney or in the male generative organs.

Etiology.—Vesical tuberculosis is most frequent in early adult life, and is more common in men. As a primary focus in the urinary system, vesical tuberculosis is decidedly rare, the infection being carried by the blood-stream. In the very great majority of cases the infection is secondary to a tuberculous focus in a kidney or in the epididymis, prostate, or

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seminal vesicles. When secondary to a renal focus, the infection is the result of direct spread along the ureter or from the infected urine, but when following a focus in the prostate or vesicles, the tuberculous process extends directly into the bladder. In cases in which both the bladder and one or both kidneys are infected, much discussion has arisen as to whether the disease has been primary in the bladder and ascended to the kidney, or whether the disease has been primary in the kidney and descended into the bladder. Whereas formerly the ascending theory was held, recent work by means of the cystoscope and the ureteric catheter has shown that the disease is almost always primary in the kidney, and that the early symptoms referable to the bladder frequently exist even while the latter remains non-infected by tuberculous disease.

Pathology.—Tuberculous disease in the bladder begins in the mucous membrane as minute greyish elevations surrounded by a zone of inflammation. They tend to caseate and break down, forming minute areas of ulceration, which coalesce to form larger ulcers, with thin, undermined edges and a red granular base. In old-standing cases the bladder-wall becomes much thickened and fibrous.

The area of infection in the bladder-wall frequently affords evidence of the source of infection. When the disease is secondary to renal tuberculosis it may be at first confined to the immediate area surrounding the ureteric orifice of the affected side; with disease of the seminal vesicle, it is found behind the trigone, and in those cases in which a prostatic focus has ruptured into the bladder, an ulcer may be found at the side of the trigone.

Symptomatology.—Gradual, but persistent, increasing frequency of micturition is the first symptom, the desire to empty the bladder being imperative at gradually decreasing intervals during both day and night. The symptom is intensified by cold or the taking of unsuitable diet, such as alcohol or highly-spiced or highly-flavoured foods. Pain is experienced if the patient attempts to hold his urine when the desire to micturate is present, and there is a burning pain in the glans penis in the male or in the external urinary meatus in the female at the end of micturition. Hæmaturia is frequent, the last few drops of urine at the end of micturition being tinged with blood. More severe hæmaturia may occur occasionally, especially when an extravescical focus ruptures into the bladder.

The urine is acid, of low specific gravity, and of a pale, hazy, or opalescent appearance from the presence of a small amount of pus.

Diagnosis.—The presence of vesical irritability in a young adult in whom venereal disease can be excluded is suggestive of tuberculous infection. The microscopic examination of the urine will show the presence of pus and blood, and careful search should be made for tubercle bacilli, which, if found, make the diagnosis conclusive. Failure to find tubercle bacilli must not be accepted as final, as repeated examinations of the centrifugalized urine may be necessary, or the inoculation of the suspected urine into a guinea-pig. A careful cystoscopic examination should be made of the bladder, preferably under an anæsthetic, when the small, discrete, greyish tubercles or distinct ulceration may be seen.

There may also be evidence of tuberculous disease in the epididymis, seminal vesicles or prostate, or the lower end of one or other ureter may be felt to be thickened per rectum or per vaginam.

Prognosis.—The course of tuberculous cystitis is gradually progressive unless adequate treatment is undertaken. When the disease is secondary to infection in one kidney, nephrectomy with ureterectomy, followed by the treatment detailed below, holds out a good promise of cure, but when it is secondary to genital tuberculosis the prospect is less favourable, owing to the difficulty in removing the primary focus of infection. The supervention of septic inflammation in the bladder increases the gravity of the case, which may end in ascending suppurative pyelonephritis.

Treatment.—In those cases in which the vesical infection is secondary to a unilateral renal tuberculosis the removal of the diseased kidney and ureter and subsequent general treatment will usually be sufficient to cure tuberculous cystitis. When, however, the renal infection is proved to be bilateral, when prostatic or vesicular disease is present, or when the disease appears to be primary in the bladder, general treatment is necessary.

General treatment includes plain and nourishing food, and avoidance of alcohol and highly-flavoured or highly-spiced articles of diet. Cod-liver oil, malt, cream, and milk should be given, and the patient should live in a dry, warm climate such as is found in Egypt, the south of France, or the Canary Islands.

For drugs, sandal-wood oil in 10-min. doses, together with belladonna and hyoscyamus, re-

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lieve the irritation of the bladder, whilst guaiacol (capsules of 5 min.) has been recommended. In the later stages of the disease, when vesical irritability is a marked symptom, morphia in the form of suppositories may be given. Urinary disinfectants may be required when septic infection is added to the tuberculous disease.

Tuberculin should be given by injection in all cases, even when the primary renal focus has been removed. In many cases great amelioration of all symptoms has followed a course of gradually increasing doses of tuberculin. If any reaction is observed by slight pyrexia, increase of frequency of micturition, or of hæmaturia, the dose should be diminished, and only again increased with caution. The treatment should begin with a dose of 1/10,000 mg. of tuberculin (T.R.), gradually increased week by week and continued for at least one year.

Local treatment is entirely contraindicated. Instrumentation should be limited to cystoscopic examination to complete the diagnosis. Irrigation of the bladder with disinfectant solutions or instillations of iodoform in paraffin have not given good results, while direct applications made to a tuberculous ulcer through a suprapubic or perineal cystotomy have not only been disappointing, but open the road to septic infection, so that the ultimate condition of the patient is rendered worse by operative interference. In addition, not infrequently a fistula infected with tuberculous material remains.

R. H. JOCELYN SWAN.

BLEPHARITIS (see EYELIDS, AFFECTIONS OF).

BLEPHAROSPASM (see EYELIDS, AFFECTIONS OF).

BLOOD, CLINICAL EXAMINATION OF (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

BLOOD-CULTURES (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

BLOOD-PRESSURE.—Of late years, in proportion as our instruments for blood-pressure measurement have developed in accuracy, arterial blood-pressure observations in clinical medicine have played a part of increasing importance in connexion with diagnosis, prognosis, and treatment.

The essential factor on which blood-pressure depends is the energy of the ventricular con-

traction of the heart, by which means the blood is thrown into the arteries. Arterial pressure is also maintained by the resistance encountered in the arteries and capillaries, owing chiefly to the partial muscular contraction (so-called tonus) of their walls, and in lesser degree to the friction of the circulating blood, due partly to its viscosity and partly to the branching of the arterial tree. Variations in arterial tonus, and hence of general blood-pressure, are dependent upon the vaso-motor mechanism. The abdominal vessels are those which by this means exercise the greatest influence upon the blood-pressure. The intermittent flow of blood in the arteries, the result of cardiac action, is converted into a constant one by the elasticity of the arteries. Besides these factors, arterial blood-pressure is also influenced to a less extent by the total volume of the circulating blood.

By *systolic pressure* is meant the maximum internal pressure to which the arterial walls are subjected at a time corresponding to the systole of the ventricle. The lowest point to which the pressure falls between each pulse-beat is known as the *diastolic pressure*, and corresponds to the diastole of the ventricle. Until comparatively recently blood-pressure measurements were, for the most part, concerned with the systolic pressure, but the value of diastolic readings is becoming increasingly manifest. It is obvious that the arterial wall is continuously subjected to this diastolic pressure. *Ceteris paribus*, it increases with the peripheral resistance, and vice versa. Further, a loss of elasticity of the arterial system will cause a diminution in diastolic pressure. Lastly, premising that the peripheral resistance remains normal, the diastolic pressure will be greater or less according as the cardiac action is rapid or slow. By *pulse-pressure* is meant the difference between systolic and diastolic readings. This is important from several points of view. For instance, it has been found that, as a rule, the amount of urine varies in proportion to the pulse-pressure.

Instruments.—In this country the instruments most generally adopted for measuring blood-pressure are those in which one of the numerous modifications of the method of *circular compression* is used. This method was devised by Riva Rocci and by Leonard Hill. It has been shown in animals to yield readings which agree with those obtained by direct

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observation by means of a manometer connected with the interior of an artery. In using an instrument constructed on this principle (Fig. 13) the arm is encircled by a rubber bag, which is covered and maintained in position by a band of canvas or other resisting material. The cavity of the bag is connected, by means of two tubes, (a) with a pump or other arrangement by means of which air can be blown into it, (b) with a manometer by means of which the pressure is recorded in terms of millimetres of mercury. When an observation is to be taken, air is pumped into the bag so as gradually to compress the limb at the site of application. The blood-vessels at the site of

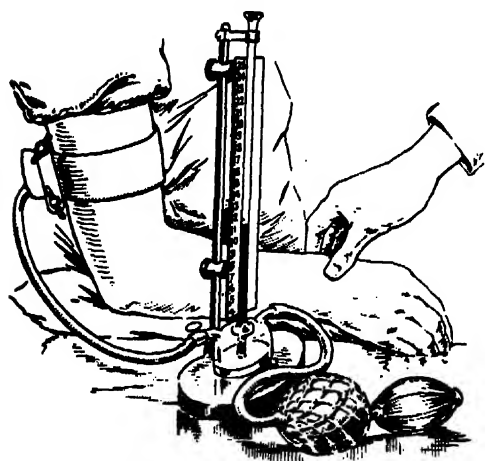


Fig. 13.—The Riva-Rocci sphygmomanometer.
(After Janeway.)

compression thus become obliterated. Compression is continued to a point slightly above that at which the pulse in the limb distally to the area of compression disappears. The pressure is then gradually reduced and manometer reading at the point at which the pulse reappears noted. The number thus recorded is the systolic pressure.

Besides this tactile means, another way of reading blood-pressure is by the so-called *auditory method* devised by Korotkow, and introduced into this country by George Oliver, to whom the profession owes many valuable instruments for the measurement of blood-pressure. In the auditory method a tambour or similar arrangement connected with the stethoscope is applied to the artery below the armlet. On reducing the pressure in the armlet a series of characteristic sounds is audible.

The appearance of a sharp clear tone denotes the first passage of blood beneath the armlet. This tone may then become indistinct and combined with a murmur. The murmur again becomes replaced by a sharp clear tone, which rather suddenly becomes weaker and duller and finally disappears. It has been shown that the point of appearance of the first sound serves as the index of systolic pressure. MacWilliam and Spencer Melvin have furnished good reasons for believing that the point at which the second clear tone becomes weaker and duller should be regarded as corresponding to the diastolic pressure.

The palpation and auditory methods, as described above, furnish probably the most reliable means that are open to the practitioner of measuring systolic pressure, and the two methods will in most cases be found to agree very approximately, the auditory reading being at least as high as, or even higher than, the tactile one. The auditory method is also recommended for the estimation of the diastolic pressure, for it furnishes a far more accurate indication of this than such means as the observation of the lowest point of maximum oscillation of the column of mercury in the manometer.

Fallacies. — With regard to the influence of the vessel-wall upon systolic blood-pressure readings, it has been shown that many cases of sclerotic and especially tonically contracted arteries, particularly in the lower extremities, yield higher readings than do normal arteries in the same patients, all precautions to avoid error being duly taken. The influence of the vessel-wall in these cases is therefore manifest. As to the effect of the superficial tissues, it has been shown that provided the breadth of the armlet bears a certain relation to the diameter of the limb compressed, the resistance of these tissues does not modify the readings in the absence of muscular contraction. To meet these conditions the breadth should not be less than 12 cm. (Edema of the limb interferes with the accuracy of the method. MacWilliam and Melvin find that conditions where there is considerable discrepancy between the auditory and tactile readings, or in which the former is lower than the latter, are unsuitable for the auditory method. These observers note that such conditions may be due to improper application of the tambour, and may be remediable.

Precautions.—Blood-pressure observations should be made at the same time of the day,

BLOOD-STAINS, EXAMINATION OF

preferably before meals and not immediately after exertion. Before taking a reading muscular relaxation must be ensured, the patient being recumbent if possible. The armlet should be at the level of the heart. Great care should be taken to obtain mental tranquillity, and the first observation should be disregarded, especially in nervous subjects.

Oliver gives 90-145 mm. of mercury as the normal systolic pressure in men, and values of 5-10 per cent. less in women. In men over 40 years of age the limits may be given as 110-160 mm. (Oliver); whilst in children under 15 the average may be taken as 100-105 mm. Daily variations exist.

High arterial pressure may result from temporary causes, such as nervous disquietude or toxæmias. Clinically it is met with in arterio-sclerosis and chronic interstitial nephritis, these conditions furnishing some of the highest readings met with; also in chronic parenchymatous and acute nephritis. Aortic insufficiency is characterized by a high systolic and a low diastolic reading, and Leonard Hill has shown that a permanent difference between the arm and leg readings (both being taken at the heart level) is a valuable sign. The present writer has shown that in thoracic aneurysm a difference of pressure on the two sides may be of diagnostic importance. High pressure occurs in marked degree in cerebral hæmorrhage, and has been described in association with the visceral crises in tabes. A sudden rise of pressure may afford useful evidence of perforation in enteric fever. Low blood-pressure occurs in wasting diseases such as pulmonary tuberculosis and cancer of the digestive system. It is typical of Addison's disease. In diphtheria a marked fall suggests a bad prognosis. The pressure is low in typhoid fever, and falls rapidly with the occurrence of hæmorrhage. Further, low pressure is of great diagnostic value in shock and collapse.

(For the treatment of high arterial pressure, see ARTERIAL DEGENERATION.)

OLIVER K. WILLIAMSON.

BLOOD-STAINS, EXAMINATION OF.

—Appropriate examination can supply a definite answer to the questions—(1) Is the stain blood? (2) If blood, did it belong to man or other species of mammal, to bird, fish, or reptile? Other questions frequently asked by counsel are—(1) How old is the stain? (2) Is it arterial or venous blood? (3) Is it menstrual blood? (4) Did the blood come

from man, woman, or child? (5) Did it come from a living or a dead body?

Absolutely fresh stains will differ from old ones, but no definite rules can be given for ascertaining the date at which an old stain was produced. Arterial blood when effused is bright scarlet in colour, while venous blood is dark red. The latter, however, when exposed to air becomes re-oxidized to the colour of arterial blood, and the dried stains cannot therefore be differentiated. When a small artery is severed the blood spurts out, and if deposited on flat surfaces the stains will frequently have the shape of an inverted soda-water bottle or exclamation marks. Venous blood is poured out in a continuous stream, and does not give rise to such appearances. When a large artery has been severed the blood-pressure is almost immediately lost, and the blood is not carried to any distance.

Further, in the majority of cases in which death is the result of hæmorrhage, the bleeding will have been both arterial and venous. Marks indicating that the blood has been sprayed will show that it has been forcibly ejected from a vessel, and therefore derived from a living body. Menstrual blood is more fluid than ordinary blood, from admixture with vaginal mucus, which may also give it an acid reaction. Microscopically, epithelium from the lining of the vagina may be detected, but, in examining dried stains on garments, it must be remembered that the garment may have been soiled by ordinary vaginal discharge before the blood-stain was produced. The question of menstrual blood will usually be raised in cases of rape, and the examiner should search for a wound from which the blood might have effused, and admit frankly his inability to differentiate menstrual from ordinary blood.

All blood-stains should be examined (1) by the naked eye and with a hand lens, (2) by chemical tests, (3) by microscopic tests, (4) by spectroscopic tests, (5) by biological tests.

1. **Examination by naked eye and with hand-lens.**—The position, number, and size of the stains should carefully be noted. The colour in recent stains is reddish, older stains being brownish red or almost black. On black or dark-coloured fabrics stains are difficult to detect, and may be more easily observed by artificial light than in daylight. On metal objects blood may appear as dark spots or smears, which later on become fissured. Cloth fabrics are stiffened owing to the coagulation of the albuminous material of the blood, the

BLOOD-STAINS, EXAMINATION OF

surface of the stain is often more or less shining, and examination with the lens shows small coagula matting together the fibres of the cloth.

Many substances produce red or brownish-red stains resembling those of blood, e.g. fruit, jam, dyestuffs, rust, faecal matter, dried solution of rubber, paint, and various chemical solutions.

For the further tests, solution of the stain is required. The colouring matter of fresh blood (oxyhæmoglobin or reduced hæmoglobin) is readily soluble in water. In older stains it is transformed into methæmoglobin and then hæmatin, both of which are less soluble in water.

In order to obtain the corpuscles as near the natural form as possible, the solvent should have the same specific gravity as the liquor sanguinis. Normal saline solution, or a solution composed of 1 part glycerin to 7 parts of distilled water, is suitable in most cases.

Cloth, leather, metal, wood, and plaster are the materials on which blood-stains are most frequently found. In the case of cloth the stain should be cut out and placed on a watch-glass, moistened with the solvent, and, if solution does not take place in a short time, covered with another glass to protect it from dust and prevent evaporation, and left for some hours. At the end of the period the fabric is gently pressed with a glass rod to squeeze out the solution.

Mordants used in dyeing may fix the stain and prevent solution. In such cases separate pieces of the fabric should be treated with liquor ammoniæ B.P. and a weak solution of citric acid: solution will usually be obtained with one or other menstruum. In the former case the colouring matter will be in the form of alkaline hæmatin, and in the latter of acid hæmatin. Ammonia is also a good solvent for old stains which are difficult of solution in other menstrua.

In dealing with wood and leather, where the stain is thick, a portion may be lifted off with a knife, or a thin shaving removed and then bent so that the surface of the stain on the convex side can be made just to touch the surface of the solvent. The tannic acid present in leather and oak fixes the stain, and when the amount of blood is very small a thin shaving bearing the stain should be digested in 2-per-cent. HCl.

When dealing with plaster the stain is scraped off and treated as in the case of cloth.

Stains on metal are best scraped off into a watch-glass and then treated with the solvent.

2. **Chemical tests.**—i. When a portion of the red solution is boiled in a test-tube the red colour disappears and a brown flocculent precipitate is obtained, if the solution is blood. This is due to coagulation of globulin, which carries down with it the colouring matter of the blood. The addition of caustic potash causes the precipitate to dissolve, and produces a dichroic solution, green by transmitted light and red by reflected light.

ii. *Ammonia test.*—A little weak solution of ammonia added to a solution of blood produces no change in colour, but causes the red juices of fruits to turn green and solutions of logwood, madder, or cochineal crimson.

iii. *Guaiacum test.*—To a small quantity of the solution add a drop of fresh tincture of guaiacum and shake gently, then pour in a little ozonic ether, when, if the solution contains blood, a blue line, varying in depth of colour with the quantity of blood, will appear at the junction of the fluids. As the colour is due to the iron-containing radical of hæmoglobin, a positive result will be obtained even after blood has been boiled, and the test will also succeed in the case of washed stains if any of the colouring matter of the blood remains. A negative result is of the utmost value in proving the absence of blood, but positive reactions must be interpreted with greater caution. Ferric salts and some other substances give a blue colour on the addition of guaiacum alone, and in such cases the test is not applicable. Bile and other substances give a positive result with guaiacum and ozonic ether after the lapse of some time, whereas with blood the reaction is obtained at once or within a few seconds. The test is a very delicate one and, as a confirmatory test, perfectly reliable. No examiner should rely on one test only, and the guaiacum test can be taken as corroborative of the microscopic or spectroscopic findings.

iv. *Hæmin crystals.*—A small portion of coagulum (or a few drops of the coloured solution) is placed on a microscope slide, a drop of glacial acetic acid added, and mixture effected by a glass rod. There is next added a small crystal of sodium chloride, a cover-glass is placed on the top of the fluid, and the slide heated above the Bunsen flame till ebullition takes place. The preparation is then allowed to cool and examined with a magnification of about 300 diameters. Hæmin

BLOOD-STAINS, EXAMINATION OF

crystals are rhombic prisms, yellowish-red to brownish-black in colour, and occur singly or in clusters. The production of hæmin crystals is indisputable evidence of blood, but failure to obtain them from true blood-stains is not uncommon.

The quantity of stain solution obtained is often so small that the guaiacum test is the only chemical test available. When there is only a little solution a few drops should be sucked up on a piece of filter paper, a drop of guaiacum superimposed, and then a little ozonic ether poured on. If blood is present the blue colour will be best developed at the margins.

3. Microscopic examination.—Success in finding corpuscles can only be looked for in the case of recent stains, and the best results are attained when a small portion of clot is available for examination. The solvent used should always have the same specific gravity as the blood. The presence of corpuscles is conclusive evidence of blood, and further information is derived from the size, shape, and presence or absence of a nucleus. In man and other mammals, with the exception of the camel tribe, the red corpuscles are circular, bi-concave, non-nucleated discs. In the camel tribe they are oval and non-nucleated, while those of birds, fishes, and reptiles are oval and nucleated. The corpuscles of man differ only slightly in average size from those of many other mammals, and the conditions which exist in medico-legal work make it impossible to rely on measurements, for when a stain has been treated with a solvent (even though it has the same specific gravity as the liquor sanguinis) it is impossible to be certain that the corpuscles have resumed their normal size. Biological tests will decide the source of the blood, and no attempt should be made to come to a conclusion from the size of the corpuscles.

4. Spectroscopic examination.—This is the most delicate test for blood. If there is a sufficient quantity of solution it should be placed in a flat glass cell and examined with a good hand spectroscope. When the amount of solution is small, use must be made of the micro-spectroscope and the solution examined in a narrow glass tube (a piece of barometer tubing to one end of which a flat glass foot has been fused is suitable). The chief spectra to be looked for are those of oxyhæmoglobin and reduced hæmoglobin in recent stains; that of methæmoglobin when the stain has been

exposed to the air for some days, and that of hæmatin in old stains.

Oxyhæmoglobin.—The spectrum shows two dark absorption bands between the Fraunhofer lines D and E, the one nearer D being about half the width of the other. After some time, varying from an hour or so in warm weather to a day or more in winter, the blood colouring matter is changed into reduced hæmoglobin.

Reduced hæmoglobin.—A single broad band appears between the lines D and E. If the solution is re-aerated by shaking up with air, the spectrum of oxyhæmoglobin reappears. A solution containing oxyhæmoglobin may be made to give the spectrum of reduced hæmoglobin by the addition of a little freshly prepared ammonium sulphide solution or Stokes's solution (dissolve a fragment of ferrous sulphate in water, add excess of potassium tartrate, then sufficient dilute ammonia to dissolve any precipitate).

Methæmoglobin.—One broad band in the red between the lines C and D and two thinner and fainter bands between D and E.

Hæmatin.—The spectrum varies with the solvent used.

Acid hæmatin.—This is obtained when the stain has been treated with citric or acetic acid. The spectrum shows a band extending from the C line midway to the D line, and a wider band between the D and E lines terminating at E.

Alkaline hæmatin is produced when the stain has been treated with ammonia. The spectrum shows a band between C and D which touches the D line.

Hæmochromogen or reduced hæmatin is obtained by adding Stokes's solution or ammonium sulphide to alkaline hæmatin. The spectrum is very characteristic, and shows two bands similar to those of oxyhæmoglobin but somewhat nearer the violet end. One band lies between D and E, and the other coincides with E_b.

Carboxyhæmoglobin.—This combination appears in the blood of those who have died from CO poisoning. The spectrum resembles that of oxyhæmoglobin, but the bands are nearer the violet end, and no reduction follows the addition of ammonium sulphide or Stokes's solution.

5. Biological test.—This test has developed out of the study of immunity, and enables us to determine the animal from which the blood was derived. The method usually employed

BLOOD-STAINS, EXAMINATION OF

is the *precipitin test* (*Uhlenhuth's test*). It depends on the precipitate which forms when a high potency immune serum is mixed with its homologous protein antigen. It is a specific protein test and not a blood test, and a positive result is obtained with other proteid substances such as albuminous urine, spermatic fluid, etc. It is therefore necessary first to prove the presence of blood by chemical, microscopic, and spectroscopic tests. The immune serums used in the tests are prepared by the intravenous or intraperitoneal injection of rabbits at intervals of five or six days with foreign defibrinated blood or serum (antigen), e.g. blood or serum of man, sheep, pig, etc. After several injections, *precipitin* (immune body) appears in the serum of the animal injected. When the serum has reached a high potency the animal is bled and the serum, after separation from the clot, stored in sealed ampoules. Such a serum will react only with the type of blood (homologous antigen) which produced it. Difficulty will only arise in differentiating the blood of closely allied species, e.g. horse and ass, sheep and goat, dog and fox. But in such cases the heterologous blood will only react in strong solutions, whereas the homologous antigen will give a positive reaction in high dilutions.

The test is performed thus: A solution of the stain is obtained with normal saline and cleared by the use of the centrifuge or filtration. If only a small quantity of solution is available the watch-glass containing it may be tilted to the side and allowed to stand for some hours. The solid matter falls to the bottom, and the supernatant fluid is usually sufficiently clear for the test. A little of the blood solution is sucked up into a capillary tube, and then a small quantity of the antiserum. A positive result is shown by the formation of a white ring at the junction of the fluids. The cloudiness should appear at once or within a few minutes. If a positive result is not obtained with human antiserum, then antbovine, antiequine, and other antisera are tried till the source of the blood is found. A solution from a portion of the unstained fabric should be tested with antiserum as a control.

For the complement-deviation test, see SEROLOGICAL DIAGNOSIS.

A. ALLISON.

BLUE DISEASE (see HEART, CONGENITAL DISEASE OF).

BOOKHART'S IMPETIGO (see IMPETIGO CONTAGIOSA).

BONE, NEW GROWTHS OF

BOIL (see FURUNCULOSIS).

BONE, ACUTE NEOROSIS OF (see OSTEOMYELITIS).

BONE, CYSTS OF.—Cysts of bone result from various pathological conditions. In young children a slowly growing myeloma usually takes the form of a cyst; it is commonest in the upper end of the humerus, but may occur in the upper end of the femur. Spontaneous fracture is, as a rule, the first symptom.

In older children and in young adults osteitis fibrosa is a cause of multiple cysts; it is a slowly progressive disease which affects the tibia or the radius most commonly, causing aching pain, tenderness, and enlargement of the bone.

In older people bone-cysts may result from osteomalacia and from osteitis deformans.

Hydatid disease may occur in bone, and causes multiple cysts distributed through the medulla.

In all varieties of bone-cysts spontaneous fracture and expansion are the prominent symptoms; pain is not, as a rule, of any severity.

Treatment is surgical, by curetting, resection, and bone-grafting, or very occasionally by amputation.

C. W. GORDON BRYAN.

BONE, INFLAMMATION OF (see PERIOSTITIS; OSTEOMYELITIS; EPIPHYSITIS).

BONE, NEW GROWTHS OF.—New growths of bone may be benign or malignant. The benign growths are enchondromata, exostoses, and myelomata; each of these is dealt with under its special heading. The malignant growths include primary sarcoma and secondary carcinoma and sarcoma.

PRIMARY SARCOMA OF BONE

Sarcomata are either purely cellular, very vascular, rapidly-growing tumours which destroy the affected bone without great enlargement, or more slowly-growing tumours containing bone, cartilage, or fibrous tissue, and giving rise to a definite enlargement of the bone in which they grow.

Sarcomata originate most commonly in the cancellous bone or the periosteum of the femur, tibia, radius, and humerus, and in the skull, jaws, vertebrae, and pelvic bones. They usually occur before the age of 30, and are more frequently seen in males than in females. In old people, however, they occur as a sequela of osteitis deformans. They may be endosteal or periosteal.

BONE, NEW GROWTHS OF

An *endosteal* sarcoma originates in cancellous bone, and causes an apparent expansion, by absorption of the inner aspect of the compact bone while a deposit of new bone takes place externally.

Symptomatology.—There is pain of a boring character, worse at night, and a localized enlargement of the end of the bone. Spontaneous fracture is sometimes the first sign of the disease. As the compact bone becomes thinned, egg-shell crackling may be detected, and when the growth has burst through the bone enclosing it, it forms a nodular swelling, often pulsating and giving a sense of fluctuation. In late stages the patient's temperature is hectic. The articular cartilage resists the growth of the tumour, and the adjacent joint does not become invaded.

A *periosteal* sarcoma arises in the deep layers of the periosteum, the superficial fibrous layer forming a pseudo-capsule to the tumour. These tumours are extremely malignant, and are liable to undergo partial ossification and formation of areas of cartilage.

The **symptoms** are pain and a swelling on the surface of the bone, spindle-shaped or pyriform if the tumour surrounds the bone or is localized to one aspect. The skin overlying the tumour is vascular, large veins being obvious, and it is hot and gives a sense of fluctuation. Hectic fever is not uncommon. In advanced stages spontaneous fracture may occur.

Diagnosis of primary sarcoma of bone.—In endosteal sarcoma, X-rays show a rarefied area in the bone, with a well-defined edge and often trabeculated; there is no surrounding sclerosis. The X-ray appearances of periosteal sarcoma are very characteristic, showing a tumour outside the compact bone, with striæ of ossification at right angles to the surface of the compact tissue.

Endosteal sarcoma must be diagnosed from myeloma, which is very slow-growing, from bone-cyst, tuberculosis, chronic abscess due to pyogenic organisms, and gunnia. Periosteal sarcoma may resemble abscess, aneurysm, subperiosteal hæmatoma from scurvy, and a Baker's cyst in connexion with osteo-arthritis.

In doubtful cases the diagnosis may be settled by X-rays, the absence of leucocytosis, exploration with a wide-bore needle, or exploratory incision; microscopic examination may be made of fragments removed.

The **prognosis** of sarcoma of bone is very bad, metastases supervening in the lungs and

lymphatic glands. The results even of high early amputation are most disappointing.

Treatment.—Radical treatment by amputation is adopted if no sign of metastases can be found by clinical examination and by X-ray examination of the lungs. Amputation must be high enough to remove the whole of the bone in which the growth originates, and the whole of all muscles in relation to the tumour; the lymphatic glands of the limb must also be taken away. This means usually, for sarcoma of the humerus, a fore-quarter amputation, for growth of the femur a hip amputation, and if a bone of the forearm or leg is affected amputation through the arm or thigh.

So frequently, however, is amputation followed by the appearance of metastases, that question arises as to the wisdom of an operation which involves a grave risk to life from shock, and whether enucleation or resection, followed by radium treatment, may not be a wiser procedure. If amputation is undertaken, every precaution must be adopted to prevent shock and hæmorrhage, by using gas-oxygen and local methods of anæsthesia, and by preliminary and after-treatment.

The results of Coley's treatment have been disappointing. In inoperable cases, and for recurrence, radium or X-ray treatment should be employed; pain must be relieved by opium, aspirin, and allied drugs.

SECONDARY MALIGNANT TUMOURS OF BONE

The organs in which **carcinoma** leads most commonly to secondary deposits in bone are the breast, the thyroid, the kidney, and the prostate. There may be a localized endosteal tumour of one bone, or a diffuse invasion of the interior of several bones—so-called *osteomalacia carcinomatosa*. Bending or spontaneous fracture of the bone occurs; occasionally the fracture subsequently unites. In the skull bones very vascular pulsating growths may occur, with a structure resembling thyroid tissue.

Carcinoma of bone usually makes its presence known by causing a spontaneous fracture, though this may be preceded by aching pain.

Treatment in most cases can only be symptomatic, but it is justifiable to excise the clavicle, if it is the site of a metastasis after the removal of the breast for scirrhus, provided there is no other sign of recurrence.

The bones of the mouth, it may be added, are frequently involved in the spread of a

BONE, SYPHILIS OF

local epithelioma, and excision of the upper jaw or resection of the lower jaw may be called for.

Sarcomatous metastases in bone are most commonly secondary to tumours of the kidney, sarcoma of the thyroid, and melanotic sarcoma. Fracture due to sarcoma does not unite.

In every case of bone tumour the possibility of its being a metastasis must be remembered, and careful search must be made for a primary focus; many cases have been recorded of the development of advanced bone metastasis before the primary focus, small and slow-growing, has made its presence known.

C. W. GORDON BRYAN.

BONE, SYPHILIS OF.—Inflammation of bone occurs in both the congenital and the acquired forms of syphilis; there may be periostitis or osteitis, diffuse or localized.

Pathology.—In periostitis the inflammation is subacute, and causes considerable hyperæmia and swelling; unless it is overcome early, a large amount of new bone is deposited, especially in children, and leads to permanent nodes. In some cases periosteal gummata are formed, and, by breaking through overlying skin or mucous membrane, allow septic infection and resultant necrosis of the bone; this sequence is commonest in the skull bones, areas of necrosis alternating with areas of dense sclerosis, the affected bone becoming heavy and in appearance "worm-eaten."

In the bones of the nose, caries with ulceration of the muco-periosteum occurs, affecting especially the vomer, ethmoids, nasal bones, and the palate, and leading to palatal perforation.

In the long bones, chronic diffuse osteo-periostitis is met with and is characterized by dense sclerosis, causing increase in thickness and weight; in children overgrowth in length also occurs, and may lead to deformity. Diffuse osteitis of the phalanges receives the name of dactylitis.

Local endosteal disease may occur in infants as epiphysitis; in adults it causes sclerosis associated with periostitis of the segment affected, and may lead to the formation of a central gumma; the sclerosis diminishes the blood supply of the affected area of bone, and, if pyogenic organisms gain access, necrosis follows.

Symptomatology. Congenital syphilis.—In the first six months of life periostitis of the skull occurs, particularly in the frontal

and parietal bones, and, if unchecked, leads to the permanent thickenings known as Parrot's nodes; from the arrangement of the swellings round the anterior fontanelle the name "hot-cross-bun skull" has been given to the condition. It is painless, and there is no inflammation of the overlying scalp. Sometimes there is craniotabes—an associated rarefaction of some areas, in which the bone is resilient and yields to pressure by the finger; but it is possible that this is rather a manifestation of coexisting rickets than of syphilis.

During the first year of life syphilitic epiphysitis is met with, and may cause defective growth and deformities; it is described under **EPHYSITIS**.

Syphilitic dactylitis is seen usually before the age of two, and most commonly affects the proximal phalanges; it is dealt with under **DACTYLITIS**.

At about the same period, affection of the nasal cartilages and bones is common and gives rise to chronic "snuffles," with muco-purulent discharge from the anterior and posterior nares; the growth of the septum is arrested, leading to depression of the bridge of the nose and a highly arched palate.

Apart from infancy, congenital manifestations occur usually about puberty, and occasionally up to the age of 25, in the form of chronic periostitis and diffuse osteo-periostitis. The former most frequently affects the tibiae, causing severe aching pain, worse at night when the patient is warm in bed, and leading to permanent nodes.

Diffuse osteo-periostitis leads to increased density, weight, thickness, and length of the diseased bone; it commonly affects the tibia, causing "scabbard tibia," and the ulna; owing to the fibula and the radius not being correspondingly increased in length, curving of the diseased bone occurs, the tibia being bowed forwards, the ulna developing a convexity inwards, with radial deflection of the hand.

All the bone lesions of congenital syphilis tend to a symmetrical distribution.

Acquired syphilis, in these enlightened days, is uncommon in bones. In the *early secondary* stage "osteocopic" pains, especially in the tibiae and skull, are caused by transient periostitis and fibrositis.

In the *late secondary* or *early tertiary* stage necrosis of the vomer, nasal bones, ethmoids, and palate may occur, leading to lachrymal obstruction and to perforation of the septum nasi and of the palate. A symmetrical peri-

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ostitis of the skull and tibiae may occur at the same stage, and will cause severe aching pain, and localized tender swellings which give an elastic sensation on palpation; the overlying skin may be hot and slightly oedematous.

In the *late tertiary* stage, periosteal gumma occurs, especially in the clavicle, sternum, ribs, and upper or lower end of the tibia. A hard swelling attached to the bone appears, with little pain or tenderness; at a later stage the centre of the swelling softens and the inflammation invades the skin; ulceration may follow, having a clean-cut undermined edge. There is often an associated perisynovial gummatosis of the adjacent joint.

Endosteal gumma occurs in tertiary syphilis, but is uncommon; there is localized thickening of the bone and œdema of the skin; slight aching pain may be present, which is most felt at night.

Diagnosis.—Attention must be given to signs of past or present manifestations of syphilis elsewhere. Of X-ray appearances, the most characteristic are density from sclerosis, and increased thickness of the compact bone from periosteal deposit. The density distinguishes syphilis from *tuberculosis*, which causes rarefaction without sclerosis; the uniformity of the density of the periosteal new bone of syphilis distinguishes it from the striæ of ossification which occur in *periosteal sarcoma*. A positive Wassermann reaction will aid in the diagnosis, but it must be remembered that in chronic syphilitic disease of bone, especially the endosteal varieties, the test often gives repeatedly negative results.

Treatment.—In the case of infants, both mother and child must be treated; to the child mercury may be given by mouth in the form of hydrargyrum cum creta, in doses of $\frac{1}{4}$ –1 gr. three times daily. Sometimes more rapid results are gained by inunction, a portion of blue ointment the size of a pea being applied to the binder, or rubbed into a different area of the skin, each night. Mercurial treatment must be continued for at least a year after clinical signs are absent, the Wassermann reaction being subsequently watched.

Salvarsan in doses of 0.01 grm. per kilo of body-weight has been used in intramuscular injections, but is not free from risk in infants, sometimes causing local necrosis or general toxæmia; at least it should not be used as a routine. A suckling child appears, however, to benefit from the administration to its mother of salvarsan and its substitutes. The mother

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should receive antisyphilitic treatment during each subsequent pregnancy.

In older children and in adults, iodide of potassium must be combined with mercury in the treatment, and intravenous salvarsan or one of its substitutes—of which galyol appears to be the most satisfactory—should be used.

Operative treatment is necessary in some cases, chiefly on account of prolonged resistance to treatment by drugs, and for the relief of chronic aching pain. For periostitis incision and curetting are used; for chronic osteo-periostitis it may be necessary to gouge away the compact bone for some distance.

C. W. GORDON BRYAN.

BONE, TUBERCULOSIS OF. Etiology.

—Tuberculosis of bone is most common in children and adolescents, and is often preceded by a slight injury. The infection of the bone by the *Bacillus tuberculosis* is usually from the blood-stream, the bacteria gaining access to the body, as a rule, through the respiratory or the alimentary tract. In some cases, however, the bones become invaded secondarily after erosion of the cartilages of a tuberculous joint, or the disease may spread from an adjacent focus in a tendon-sheath or bursa, or from a gland abscess.

Pathology.—In periostitis the deeper layers of the periosteum are infected, and the disease may spread to the underlying bone or may cause an abscess of the soft parts. Periostitis most commonly affects the *ribs*, *sternum*, and *vertebræ*, and more rarely is met with in the *tibia*.

Cancellous osteitis is the commonest form of bone tuberculosis, and occurs in the ends of the *long bones*—in children in the end of the diaphysis close to the epiphyseal line, in adults under the articular cartilage; it is usually associated with tuberculous arthritis. In children the disease occasionally begins in the epiphysis.

In the *metacarpus*, *metatarsus*, and *phalanges* a tuberculous form of dactylitis is met with. Cancellous osteitis of the *vertebræ* is considered under SPINAL CARIES.

In the *tarsus*, tuberculous osteitis usually begins in the *os calcis*, the *astragalus*, or the *cuboid*; by spreading it leads to infection of the synovial membranes of the *intertarsal*, *tarso-metatarsal*, and *ankle-joints*, and the *tendon-sheaths* of the foot.

In adults, tuberculous osteomyelitis is met with occasionally.

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In cancellous osteitis enlargement of the cancellous spaces leads to caries. In the commonest type, caseation ensues and an abscess forms inside the bone or under the periosteum; an adjacent joint becomes involved by spread under the periosteum, or by destruction of the articular cartilage.

In caries sicca the disease progresses without caseation; it most commonly affects the upper end of the *humerus* in young adults; the bone is rarefied and absorbed without pronounced symptoms, and the disease may quietly progress to ankylosis of the shoulder and natural cure, usually after about two years.

The name caries fungosa is given to a condition in which the disease has spread to the skin and the action of pyogenic organisms is added to that of the tubercle bacillus; necrosis with the formation of minute sequestra occurs, a sinus or ulcer with fungating granulations leading down to the diseased bone.

Symptomatology.—In tuberculous periostitis a chronic swelling appears, and there is increased heat of the overlying skin. Pain is of an aching character, worst at night, but in some cases nothing is noticed until a fluctuant swelling due to a cold abscess appears.

If the disease affects a *rib* a fusiform swelling of the bone appears, and increases in size as the abscess bursts through the periosteum on the superficial or deep aspect of the bone; in the latter case it forms a swelling of the intercostal spaces above and below the rib affected. In some cases a bilocular abscess is formed, its superficial part presenting over an intercostal space, its deep part, which is usually the larger, being situated between the chest-wall and the pleura. In other cases an abscess developing deep to the ribs tracks forwards or downwards and presents at the margin of the thoracic wall.

In periostitis of the *sternum* a cold abscess may form superficially and cause a fluctuant swelling in the middle line, over which the skin becomes red and thinned; or an abscess may arise behind the bone and track backwards into the mediastinum, pointing eventually at the side of the xiphisternum.

Periostitis of the upper part of the subcutaneous surface of the *tibia* is sometimes seen, and causes a fluctuant tender swelling under the skin.

Periostitis of the *vertebrae* affects their anterior aspect and causes mediastinal, lumbar, and psoas abscesses; the condition is described under SPINAL CARIES.

Cancellous osteitis most commonly occurs at the ends of the long bones; it is dealt with under ARTHRITIS, TUBERCULOUS.

In the *tarsus* the caries, beginning in one bone, soon spreads to others, and to the complicated series of synovial membranes of the foot; there is severe pain with limping, and the muscles of the calf are wasted; the foot becomes hot and swollen, and from rigidity of the intertarsal joints the movements of inversion and eversion are lost; great pain is elicited by compression of the foot. Disease of the *astragalus* leads to infection of the ankle-joint, which is kept in a position of plantar flexion. In the later stages abscesses form and break through the skin, and the entrance of secondary infection leads to hectic temperature and rapid progress of the disease.

Diagnosis.—The various lesions of bone tuberculosis must be diagnosed from similar inflammations due to other organisms. *Typhoid osteitis*, which commonly affects ribs and tibia, is distinguished by the history and by a positive Widal reaction; in *Brodie's abscesses* there is thickening of the compact bone, with subperiosteal ossification; *syphilis* causes painless dense thickenings of bones, which are not tender. But it is on X-rays that most reliance is to be placed, and they should be employed in all cases; the characteristic of tuberculous caries is diffuse rarefaction of cancellous bone with a clear area at the site of caseation, but without the peripheral sclerosis seen in a Brodie's abscess, and without sequestrum formation.

Prognosis.—In tuberculous periostitis of the *sternum*, *ribs*, and *tibia* the prognosis, under suitable treatment, is good.

Cancellous osteitis may become quiescent, and eventually cured, without spreading outside the bone, but in most cases an abscess forms and a neighbouring joint is involved. In children the prognosis is much better than in adults, but in disease of the *tarsus* at any age the outlook is always poor, a stiff and useless foot being the usual result if amputation is avoided.

In all varieties of bone tuberculosis in old people the prognosis is bad, and it is fortunate that they are comparatively rarely affected.

Treatment.—The general treatment of tuberculosis is carried out, and all foci of toxæmia such as septic teeth and tonsils should be searched for and eradicated.

In periostitis of a *rib* it is often possible to remove the segment diseased without opening the abscess; in more advanced cases the abscess must be opened and the segment of

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rib which is denuded of periosteum resected, the skin incision being closed without drainage. The abscess cavity is likely to refill, and must be emptied by aspirations.

In periostitis of the *sternum* a cold abscess may be treated by repeated aspiration, but if it progresses in spite of this it should be opened and the bone curetted.

Periostitis of the *tibia* is dealt with in a similar way, combined with splinting. For a child over 6 years a Thomas knee-splint may be used; at earlier stages a plaster-of-paris splint, with the knee extended and the ankle at right angles, should be used, a window being cut opposite the diseased bone.

The treatment of cancellous osteitis of the ends of the *long bones* is usually that of the coexistent arthritis. It is sometimes possible, however, by operation to eradicate the bone focus before the joint is affected.

Conservative treatment of tuberculosis of the *tarsus* consists in immobilization of the foot by plaster-of-paris in a position of varus, with the ankle slightly dorsiflexed. No weight must be borne on the foot, but crutches, a Thomas knee-splint, or a "peg leg" must be provided. In some early cases it is possible to hasten cure by excising the diseased bone, particularly if it is the astragalus that is affected. As a rule, however, the disease in the most favourable circumstances only becomes quiescent after years of treatment, and is very apt to light up again at any time; in many cases it slowly progresses in spite of careful treatment, becoming more and more painful and exhausting. Even when a cure with ankylosis of the tarsal joint results, the patient is considerably disabled and the foot is liable to sprains and constant attacks of oedema; flat-foot is another common sequela. In view, therefore, of the bad prognosis, a very poor foot being the best that can be expected, amputation is in many cases the best treatment and should not be delayed unduly. A Syme amputation or one through the middle of the leg will be necessary. In adults especially amputation must often be resorted to, providing as it does the only method of enabling the patient to become a useful member of society and a wage-earner.

C. W. GORDON BRYAN.

BOTULISM.—A form of food-poisoning due to *Bacillus botulinus*. Though originally described as the result of partaking of raw or undercooked sausages or ham, several cases have been observed, notably in the United

States, in which the victim had eaten fruit or vegetables preserved at home. It is noteworthy that in every case the canned or bottled food had not been cooked again before being brought to table. Experiments have shown that, if this be done, all risk from infection by the bacillus is removed.

Pathology.—The chief lesions found after death have been in the central nervous system, and include meningeal congestion and thrombosis and a hæmorrhagic form of polioencephalitis affecting particularly the walls of the third ventricle, the grey matter about the aqueduct of Sylvius, and the floor of the fourth ventricle. Visceral congestion and pulmonary infarction also occur.

The **symptoms** are usually gastro-intestinal and nervous, the latter being the more prominent. Perhaps within twenty-four hours after taking the poisonous meal there are nausea, giddiness, vomiting and diarrhoea, sometimes accompanied by fever. Cramp-like pain in the legs may be complained of, but abdominal pain is often absent. At this stage nervous symptoms may be slight or lacking, but before many hours they dominate the picture. They are chiefly referable to involvement of the cranial nerve nuclei situated in the crura, pons, and medulla. The third nerve is often affected early, and a combination of dilated pupils, dry throat, and active delirium may mimic belladonna poisoning. Facial palsy, deafness, and vagal paralysis may occur contemporaneously or be added in quick succession. These, together with other ocular palsies and weakness or paralysis of the limbs, constitute the chief symptoms. Other cases more nearly resemble acute bulbar paralysis, while instead of delirium the mental state may remain unchanged, or there may be drowsiness. The disease is often fatal within a few days, from paralysis of the cardiac or respiratory centres, but the patient may recover after a severe illness.

Diagnosis.—As well as from belladonna poisoning, botulism must be distinguished from encephalitis lethargica (q.v.), which it resembles closely, and from encephalitis of other forms.

The **treatment** consists chiefly in prevention by the efficient cooking of all home-preserved fruit and vegetables, and of sausages and ham before use. When the disease is manifest the obvious indication is by elimination to prevent further intoxication from the bowel, but by this time irrevocable damage may have been done.

FREDERICK LANGMEAD.

BRANCHIAL CYSTS

BRACHIAL NEURITIS (*see SPINAL NERVES, LESIONS OF*).

BRACHIAL PALSY (*see SPINAL NERVES, LESIONS OF*).

BRADYCARDIA (*see HEART-BEAT, ABNORMALITIES OF; HEART-BLOCK*).

BRAIN (*see CEREBRAL*).

BRANCHIAL CYSTS.—Derived from the branchial clefts, these cysts have a thin wall lined by ciliated or stratified squamous epithelium and contain turbid fluid. Most commonly they are closed, but they may communicate with the pharynx by a track passing between the internal and external carotid arteries, or they may have an opening externally in front of the lower end of the sterno-mastoid.

Signs.—A cystic smooth swelling is present in the anterior triangle of the neck, often overlapped by the sterno-mastoid; it gradually increases, and may be as big as an orange. It is freely movable in all directions, unless it becomes infected, when fixation occurs. If suppuration takes place, the skin becomes red and may ulcerate, causing a fistula which does not heal spontaneously.

Diagnosis.—The absence of lobulation distinguishes a branchial cyst from a breaking-down *tuberculous gland*, which will always have smaller glands related to it. An *epitheliomatous gland* is characterized by hardness and fixation; the primary focus is discoverable. *Primary carcinoma* may arise in branchial epithelium and form a hard fixed tumour.

Treatment.—The most satisfactory treatment is removal by dissection; this is carried out with moderate ease if there has been no inflammation and if there is no communication with the pharynx. It is possible to destroy the lining membrane by electrolysis, but this method is tedious and uncertain.

C. W. GORDON BRYAN.

BREAST, AFFECTIONS OF.—In this article are included—

1. ANOMALIES OF DEVELOPMENT.
2. MASTITIS AND MAMMARY ABSCESS.
3. TUBERCULOSIS.
4. ACTINOMYCOSIS.
5. SYPHILIS.
6. CYSTS.
7. INNOCENT TUMOURS.
8. MALIGNANT TUMOURS.
9. DISEASES OF THE NIPPLE.

BREAST, AFFECTIONS OF

1. ANOMALIES OF DEVELOPMENT

Amastia.—In *complete amastia* the mammary elevation and the nipple up-growth both entirely fail to appear; in *incomplete amastia* the nipple, with or without an areola and galactophorous ducts, is present, but the gland proper is absent. Both breasts may be lacking, or one may be well formed and capable of functional activity. Occasionally amastia is associated with faulty development of muscles or bones of the thoracic wall.

Athelia, or absence of the nipple, very rarely occurs without amastia; the corresponding areola may or may not be well formed.

In **Polymastia** a condition similar to that seen in many lower animals is found. Supernumerary breasts may be found anywhere on the line from axilla to groin, or even sometimes in the perineum or the upper and inner aspect of the thigh; occasionally a single median mamma is seen near the umbilicus, probably as the result of fusion of the two mammary lines below. Supernumerary breasts rarely exceed one or two in number; they usually possess nipples, are more common in males than in females, and are seldom capable of physiological function. Accessory nipples (*polythelia*) may be present on supernumerary breasts or on an otherwise normal mamma, either on or beyond the areola of its first nipple; when there are two adequately developed nipples on a breast, both drain the whole gland.

Diagnosis.—An accessory breast without a nipple or areola may be confused with (a) a subcutaneous lipoma; (b) an aberrant lobule attached to its parent, the normal breast, perhaps only by a thin pedicle; or (c) an axillary false accessory mamma. This last is a rare swelling firmly attached to the skin of the axilla and developed during pregnancy and lactation from hypertrophied sebaceous glands. Although such a false mamma has no nipple, it has been possible in a few recorded cases to express a milky fluid from the pores of its surface.

Treatment.—A supernumerary breast should be excised if it is chronically inflamed, or if by its location in axilla or groin it produces intertrigo or hinders the free play of the limb. If it has undergone malignant changes, it must be radically removed, with a wide area of adjacent muscles, fasciæ, and glands.

Gynecomastia is a condition in which the male breast, instead of remaining in the stage

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reached soon after birth, continues to develop until it presents the characters of the female virgin breast. Gynecomastia may affect one or both breasts; although it may occur in men otherwise well formed, it is not infrequently associated with faults in genital development and with the possession of feminine characteristics of figure and voice; it has sometimes followed castration. If the gynecomastic breast is the seat of mastitis or of carcinoma, it should be removed according to the rules to be followed for similar conditions in the female.

In **Mammary Atrophy** the breasts are small and functionally incompetent. The condition may be associated with cretinism, infantilism, or general tuberculosis, or may follow mastitis, or mumps, but in many cases no adequate explanation is forthcoming.

In **Diffuse Hypertrophy** of the breasts all the mammary constituents, but especially the connective tissue, become thickened and hypertrophied in various degrees. It may arise in association with puberty or with pregnancy. In the adolescent girl the overgrowth almost wholly occurs in the interstitial connective tissue, and shows no tendency to cessation or retrogression; in the gravid woman, on the other hand, the hypertrophy also affects the glandular elements, and tends to halt or even to recede after the completion of the pregnancy. The enlarged breasts are physiologically incompetent. The condition is nearly always bilateral, and equally obvious on the two sides; this fact may serve to distinguish it from the marked general enlargement, always unilateral, sometimes associated with a deeply placed, large, and perhaps diffuse fibro-adenoma.

Clinical features.—At first the patient presents merely an unusually Junoesque figure, but the mammary enlargement soon becomes excessive, and the heavy breasts may, in the sitting position, even rest on the thighs. The nipple becomes indrawn and the surrounding skin coarse, congested, and sometimes cedematous. The consistency is at first firm, but later it may in places become soft and jelly-like. Discomfort, a sense of dragging weight, and perhaps neuralgic pain increase, and are followed by dyspnoea, insomnia, dyspepsia, general ill-health, and perhaps by kyphosis.

Treatment.—The breasts should be supported by bandages or by strapping, general rest prescribed, and menstrual anomalies treated. In the adolescent, little improvement is to be expected from these means, but in the pregnant

woman they may palliate symptoms until the birth of the child, when some retrogression may be hoped for. Usually, especially in the rapid adolescent type, the progressing hypertrophy and its consequences eventually demand separate amputations of both breasts.

2. MASTITIS

Acute Mastitis. **Etiology.**—The active cause is the ingress of micro-organisms, especially *Staphylococcus pyogenes aureus*, and less often *Streptococcus pyogenes* or *Staphylococcus albus*. The infection most often enters along the ducts or their walls, and therefore the resulting inflammation is lobular in distribution; sometimes, however, it travels by the blood-stream or the lymphatics. The predisposing causes are considered in the succeeding paragraphs.

Varieties.—It is convenient to describe acute mastitis under several closely allied clinical types.

(a) **Mastitis Neonatorum.**—In children of either sex, within three or four days of birth, it is common to find some tenderness, granular swelling, and even redness of one or both breasts. This condition, if undisturbed, usually rapidly clears up; but occasionally, as the result of chafing by clothing or unjustifiable rubbing by meddlesome nurses, it may proceed to definite inflammation or even to suppuration. The child frets, resents interference with the breast, and may be obviously out of health; the breast is red, tumid, and very tender. Resolution may occur, or suppuration follow with its usual local signs and with fever, increased malaise, and fretfulness.

(b) **Mastitis Adolescentium.**—At puberty, in either sex, the mamma is often congested, tender, and the object of solicitude on the part of the patient. The ingress of organisms from the nipple or elsewhere at this stage determines the onset of an acute mastitis which may proceed to abscess formation (see below).

(c) **Mastitis Gravidarum.**—During the last two or three months of pregnancy microbial invasion may readily induce mastitis in the rapidly developing breast. This variety and that associated with lactation are especially liable to suppurative changes.

(d) **The Mastitis of Lactation.**—In this, the commonest variety of acute mastitis, two new etiological factors are introduced, namely, milk engorgement, and abrasions of the nipple due to injury by the child or to maceration, acous-

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panied by inadequate drying and cleansing after suckling. If one or more ducts become blocked by epithelial debris, stagnant milk is pent up in the corresponding lobes, and forms an admirable culture medium for microbial multiplication; if now organisms secure admission from a damaged nipple along the ducts or their neighbouring lymph-channels or, more rarely, from a distant focus of infection via the blood-stream, there are present all the conditions necessary for the development of acute inflammation and abscess.

Acute mastitis is commonest in primiparae, especially during the first few weeks of lactation, but is also frequent towards the end of the nursing period. Death of the child or other cause of premature and sudden cessation of suckling is a strong predisposing factor.

(e) *Apart from puberty and child-bearing*, mastitis is not very common in women. It may, however, arise in association with general pyæmia or with enteric fever, or by direct spread of infection from adjacent structures, such as the pleura, ribs, or retromammary connective tissue. Moreover, the possibility that a retromammary abscess may be secondary to disease of the spine, the pus being guided from the vertebra along the corresponding intercostal space, must be remembered.

A form of subacute mastitis, analogous to the orchitis of males, may arise in mumps, usually after the disappearance of the parotid enlargement; it rarely induces suppuration, though it may lead to subsequent partial mammary atrophy.

Clinical features of acute mastitis.—A wedge-shaped sector including one or more mammary lobes becomes tender, swollen, hard, and nodular. Malaise, pain, and fever (even up to 104° F.) ensue in degrees varying with the acuteness of the inflammatory process. The axillary glands become palpably enlarged and tender. Resolution may occur; or the part may begin to throb and become reddened and exquisitely sensitive to touch. When these signs develop, a mammary abscess should be strongly suspected (*see below*).

Prognosis.—Under careful treatment many cases, especially those of the infantile and adolescent forms, resolve, leaving no sequelæ or only a localized patch of chronic induration. Others, especially those of the pyæmic variety, or those occurring in nursing women, advance to suppuration.

Treatment.—By both local and general means rest should be secured and resistance to organ-

isms stimulated. The child should be weaned, the breast emptied with the breast-pump, and cessation of lactation and of pain encouraged by the use of belladonna fomentations. The arm should be fixed to the side and the breast supported by a bandage. Tissue resistance and reparative processes may be assisted by hot fomentations, and by passive congestion secured by the application of a Bier's bell-cup for from twenty to forty minutes daily. The diameter of the bell should be slightly less than that of the breast, and the vacuum induced in it just sufficient to cause *painless*, reddish-blue engorgement; the best results are attained by alternating five-minute periods of exhaustion and release. Should pus have already formed, neither fomentations nor congestion are to be continued without incision and thorough evacuation of all the pockets of the abscess cavity. Simultaneously, the general health must be improved by an ample digestible diet, fresh air, tonics, and the administration of grey powder. When the mastitis has become subacute or chronic the breast should be strapped, or dressed with a Scott's dressing, and probably a course of vaccines instituted.

Mammary Abscess.—The varieties of mammary abscess are:

(a) **INTRAMAMMARY ABSCESS.**—Suppuration arising within the anatomical limits of the breast itself is usually merely a further stage of acute mastitis; occasionally, it may begin in a hæmatoma infected with pyogenic organisms. The pain of the mastitis becomes more acute and often throbbing in character, the tenderness is exaggerated, and the fever, malaise, and appearance of illness increase. The breast becomes œdematous, its skin over the inflamed lobes reddened and then bluish, and the abscess, if unrelieved, eventually points and bursts, perhaps in several places. Diagnosis should never be delayed until fluctuation is obvious, for, owing partly to the depth at which the suppuration originates and partly to the mobility of the part, this sign often cannot be elicited satisfactorily until considerable local disintegration and general ill-health have supervened.

Diagnosis.—Suppuration must be diagnosed at the earliest possible moment. Progressive exaggeration of symptoms and signs and the steady increase of a polymorphonuclear leucocytosis will usually prevent mistake; in case of serious doubt the diagnosis should be cleared up by exploratory incision. The danger of overlooking the presence of pus is greater than

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that of incising an acutely inflamed breast in which suppuration has not yet occurred.

Prognosis.—If the abscess be recognized early and treated efficiently, the outlook is good as regards both the general health and the functional capacity of all the breast except the involved lobes. Neglect may lead to serious and even dangerous ill-health and to the survival of a useless, sinus-riddled mass of inflammatory scar tissue replacing the breast; in feeble women, gangrene of the breast or cellulitis of the body-wall may develop.

Treatment.—The abscess must at once be opened and drained by one or more free incisions radiating from the nipple and therefore parallel with the ducts. To ensure complete evacuation and drainage all septa in the cavity should be broken down with the finger, large drainage-tubes, not gauze plugs, should be inserted and hot fomentations applied. At the same time, every effort should be made to improve the general health by attention to the bowels, by generous diet, and by fresh air and tonics. If the abscess be small and of recent development, a cure can often be obtained by a relatively small incision combined with subsequent use of Bier's congestion cups. In chronic cases in which the abscess has been neglected and has converted the breast into a sinus-riddled relic, attempts may be made to secure healing by the use of autogenous vaccines against the predominant organisms, and by daily injection of the sinuses, through a sterilized syringe, with such a preparation as the following:

- ℞ Bism. subnit. partes vi.
- Calomel pars i.
- Ol. oliv. partes ii.
- Lanolini partes xii.

But in most cases it is wiser to remove the whole breast, which is useless and only a focus of chronic toxic absorption.

(b) **SUPRAMAMMARY OR SUBAREOLAR ABSCESS.**—A small, relatively unimportant subcutaneous collection of pus is obvious under the areola and is readily cured by incision. It occasionally originates in a Montgomery's follicle.

(c) **RETROMAMMARY ABSCESS.**—Infection of the retromammary connective tissue is apt to cause a voluminous collection of pus, upon which the mamma floats as on a water cushion. The whole breast is unduly prominent, but the overlying skin is unaltered except perhaps in the submammary groove, where the abscess

tends to point. Constitutional symptoms may be severe. Among the commoner causes of such a retromammary abscess may be mentioned: (a) escape of infective material from an intramammary abscess inwards towards the chest-wall; (b) infection of a retromammary hæmatoma; (c) perforation of the thoracic parietes by a neglected empyema; (d) costal osteo-myelitis, tuberculosis, or typhoid peritonitis; (e) passage of pus from a carious vertebra along a rib space and thence into the retromammary tissue.

Treatment.—The abscess must be opened widely, preferably through the outer half of the submammary groove, and its cause discovered. If a channel be found leading into an intramammary cavity, the communication must be enlarged and all septa broken down to ensure free drainage. Efficient drainage of an empyema must be established; a diseased rib must be excised or scraped. If the abscess contains pure tuberculous "pus" it should be evacuated, swabbed out with iodoform-petroleum emulsion (iodoform and lanoline 1 dr. each, ol. petrolei to 1 oz.), and then closed with deep sutures of readily absorbable and sterile catgut followed by Michel's suture clips for the skin; a little of the emulsion may be left in the cavity.¹ Drainage should only be resorted to if the closed method of treatment persistently fails. In all other cases, free drainage with tubes, not with gauze plugs, is necessary. Careful dressing and judicious progressive shortening of the tubes will lead to cure if the original focus of disease has been removed. If a sinus results, its mouth should be dilated and daily injections of bismuth and calomel paste given, or the whole sinus must be excised. Measures must be taken to improve the general health, and attention paid to spinal disease, if present.

Chronic Mastitis.—This disease, so common in women approaching the climacteric, derives its chief importance from its relationship to cancer. There is much evidence that chronic mastitis may be a precancerous condition; moreover, not only is it sometimes difficult to distinguish a carcinoma from some examples of mastitis, especially those in which tense cysts are included, but a malignant tumour may lie for some time concealed in an inflammatory mass.

¹ N.B.—In any case where iodoform-petroleum emulsion has been used, one subsequent aspiration may be necessary to remove reaccumulated fluid which, however, is now usually clear and quite sterile.

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Etiology.—Among the causes that may be operative are: (a) incomplete resolution of a previous acute mastitis; (b) blockage of a duct or ducts by scar tissue, by epithelial debris, or by a tumour such as a duct papilloma or a fibro-adenoma; (c) imperfect involution after lactation; (d) possibly a blow or a series of injuries; (e) the incursion of micro-organisms of debased vitality; (f) age: the disease is commonest just before the menopause, but may appear in any adult woman, whether parous or nulliparous.

Morbid anatomy.—The disease, unlike carcinoma, respects anatomical barriers and therefore, if affecting only part of the breast, shows a wedge-shaped outline. The diseased part cuts with a tough rubbery consistence, leaving a yellowish-white cut surface, occasionally with a pinkish tinge. The lobular epithelium proliferates; all the connective tissue, interacinous and interlobular, becomes thickened, unduly cellular, and infiltrated with small round cells; the lobule is increased in size and its acini in number ("hypertrophic type"). Later, fibrosis occurs, strangling many acini and much of the fatty tissue, and the affected lobes appear smaller than normal ("atrophic type").

Sometimes in such breasts small retention cysts are formed, bounded by strong sclerosed tissue and containing clear or brownish fluid. Occasionally the abundance and obvious size of such cysts justify the name *chronic polycystic disease of the breast*. When the process has advanced to this degree, both breasts are often affected, and the possibility of the development of carcinoma near one of the cysts must seriously be contemplated.

The relationship of chronic mastitis to tumours is important. A duct papilloma or a fibro-adenoma blocking a duct may induce chronic mastitis of the corresponding lobules; or carcinoma may develop in an inflammatory mass and enhance the diagnostic difficulties, especially if tense cysts be present.

Clinical features.—The attention of the patient, who is usually within a few years of the menopause, may be attracted by pain or, very occasionally, by a thin, scanty serous discharge from the nipple, but most often by the accidental discovery of granular induration in the breast. Pain, when present, may be sharp and lancinating, or dull and aching; menstruation or movements of the pectoral muscles may cause exacerbations. In the hypertrophic type the breast is often enlarged;

in the atrophic, it may be shrunken and flattened or even concave. Large cysts, if present, may be seen as bossy swellings. The whole breast may be affected, or only a wedge corresponding to one or more lobes; the superior external quadrant and axillary tail are especially liable. The disease is frequently bilateral, although not symmetrically advanced. When grasped in the fingers the affected part feels tough, leathery, and granular, but if no tense cysts are present this sensation is lost when the flat palm is substituted for the palpating finger-tips. Cysts can be detected with both flat hand and finger-grasp as rounded swellings of a consistence varying with their tenseness, from obvious fluctuation to elasticity so firm as to be almost indistinguishable from hardness. The axillary glands are frequently enlarged, tender, and firm, but lack the hardness of carcinoma; the nipple is rarely, if ever, retracted, nor is the skin adherent to the breast in chronic mastitis uncomplicated by carcinoma. In *chronic polycystic disease* cystic changes predominate, commonly in both breasts; the mammary elements are widely supplanted by many cysts separated only by fibrous tissue. Clinically many knobby swellings of different sizes and degrees of elasticity can be felt, and some of the larger cysts may be obvious on inspection.

Course.—Little change may occur for years, or atrophic processes may alter the breast into a shrivelled, flattened, indurated fibrous cake. In some cases, however, chronic polycystic disease results, or carcinoma arises within the inflammatory mass.

Diagnosis.—The diagnosis between chronic mastitis and carcinoma and the early recognition of a cancerous lump in an inflammatory mass are of supreme importance. Points of value in the differential diagnosis of these conditions are mentioned on pp. 191 2.

Treatment.—If there be no suspicion of carcinomatous change, the breast should be protected from friction or injury and supported by a firm broad doanett bandage or by strapping. A Scott's dressing, replaced every three or four days, is often of great value by causing gentle counter-irritation as well as pressure and support. It is made by smearing ung. hydrargyri camph. on strips of lint, placing them in an imbricated manner on the breast, and fixing them with firmly and evenly applied strips of plaster. Later, massage with lin. saponis cum pot. iod. or with mercurial ointment, or painting the part with iodine, will

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often hasten resolution. If pain be a prominent symptom, belladonna ointment or paint will, with support, usually give relief.

If, however, polycystic disease be present, or if the age of the patient or the clinical signs cause the slightest suspicion of carcinoma, operative measures should be adopted. After exploratory incision to determine the absence of obvious cancer, the affected part, usually the whole breast, should be excised; it should then be microscopically searched for carcinoma in order that, if that complication be found, the radical operation may be performed without delay.

3. TUBERCULOSIS

(a) **Miliary Tuberculosis** of the mamma is merely an expression of the general disease and is of no special surgical interest.

(b) **Diffuse Tuberculosis** of the breast may arise by infection from adjacent tissues or from the blood-stream. It is practically always unilateral. One or more indurated areas can be felt both with the flat palm and with the finger-tips; local adhesions to underlying fascia and to skin occur, sometimes even causing an orange-rind appearance. Central softening follows, the skin becomes discoloured and gives way at many points; the breast is then found to be a useless mass riddled with sinuses which are lined with feeble granulation tissue and discharge a thin watery "pus." The axillary glands are frequently enlarged and matted together.

(c) **Chronic Encysted Abscess.**—Occasionally the local surrounding sclerosis is strong enough to wall-in a tuberculous centre and to prevent its extension through the breast or to the surface. A lump is then palpable, merged in the breast, with adhesions to the skin; it may be quite firm and non-fluctuant, though often it is more elastic near its centre. The axillary glands are usually enlarged. Similar encysted abscesses may be due to other microbes such as the staphylococcus.

Treatment.—If an encysted abscess be present, or if diffuse tuberculosis of the breast be diagnosed early while the purulent collections are few and distinct, arrest of the disease can often be secured by aseptically incising and evacuating the abscesses, gently cleansing their walls with gauze, partially filling them with an iodoform-petroleum emulsion, and hermetically closing them with suture clips. In still earlier cases improvement may follow aspiration of abscesses combined with the use of Bier's congestion cups. If sinuses are few in number

they may be curetted; but usually the most satisfactory treatment for all cases in which sinuses are present is excision of the breast (without the pectoral muscles) and of any affected axillary glands.

(d) **Tuberculous Retromammary Abscess** is considered under Mammary Abscess, p. 185.

4. MAMMARY ACTINOMYCOSIS

This condition is rare. The streptothrix may reach the breast directly from a local skin abrasion or from diseased lung and pleura; possibly sometimes it is blood-borne. It causes either a local granulomatous nodule surrounded by much fibrous tissue or, more often, a diffuse induration of the breast, which soon softens at several points. The overlying skin becomes purplish and gives way to allow the escape through numerous apertures of a viscid fluid containing "pepper granules"; this fluid is at first clear, but secondary pyogenic infection soon induces a purulent character; the axillary glands show no change until mixed infection occurs, but may then become enlarged. Healing with distorted nodular scar tissue may occur at the older sites, while spreading continues through the connective-tissue planes elsewhere.

If pepper granules cannot be found, diagnosis by culture may be necessary; it must then be remembered that the growth may not appear for some weeks, and that some families of the streptothrix grow best under aerobic, others under anaerobic conditions.

Treatment.—The sinuses may be curetted; but removal of the breast is more satisfactory. At the same time massive doses of potassium iodide (40–90 gr. thrice daily) should be given.

5. SYPHILIS

The secondary rashes and condylomata that may appear on the mammary skin are discussed under SYPHILIS.

Chancre of the Nipple.—This condition is now comparatively rare, but may be found in a normal wet-nurse suckling a syphilitic child, or even in any woman with a braded breast who allows the baby's mucous tubercles to come into contact with it. The mother herself is almost always immune. The sore may conform to any of the following types:

(a) A mere fissure or excoriation which persists, but without surrounding induration.

(b) A small, intractable, rounded, slightly

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raised reddened area of skin with surrounding and subcutaneous induration.

(c) A definite ulcer, which may be a later stage of (a) or (b). It presents a dirty, sloughy or scabby surface, with scanty discharge; it is usually shallow, and in the centre of a flat, raised, hardened surface.

(d) Occasionally pyogenic infection occurs, and phagedenic spread and sloughing may then be extensive.

(e) A true Hunterian chancre is comparatively rare in this situation.

The local "sore" is often insignificant and readily overlooked. It is usually painless; it is commonly placed on the areola or on the neighbouring skin; the actual nipple frequently escapes but may be swollen. Multiple sores of simultaneous origin may sometimes be seen, and the condition may be bilateral. The axillary glands are enlarged and hard, but mobile and not tender.

Diagnosis is made by the appearance and persistence of the sore and by the character of the axillary glands; it is confirmed by a history of exposure to infection, by the discovery of the *Spirillum pallidum* in scrapings, and later by the appearance of secondary signs of generalized syphilis and by the presence of a positive Wassermann reaction.

The local condition must be differentiated from:

(1) A vaccine sore, infected from the baby's arm. Such sore runs the usual vaccinal course and the glands are tender and not so discrete.

(2) Paget's disease of the nipple (*see p. 193*). The chancre develops more rapidly, is of a duller-red colour, and causes less discharge.

Treatment is that appropriate for general syphilis, namely novarsenobillon and mercury. Locally, the sore should be frequently washed clean, dried, and then dusted with a powder composed of equal parts of calomel, zinc oxide, and starch.

Tertiary Syphilis of the Breast is uncommon; it may take the form of a localized gumma or of a diffuse gummatous infiltration. A gumma is a readily palpable rounded mass; as it enlarges it adheres to the skin, which may become pitted like orange-rind. The integument becomes purplish and gives way with a gush of pus, leaving a deep ulcer with a sloughy (wash-leather) base. **Diagnosis** is then easy; in the earlier stages and in the diffuse type it can be aided by application of the Wassermann test and by

the discovery of other clinical evidences of syphilis.

Treatment.—Novarsenobillon, mercury, and iodides should be administered according to the rules laid down in **SYPHILIS**.

6. MAMMARY CYSTS

The clinical characters of a mammary cyst vary somewhat with its nature and with its tenseness. Generally an innocent cyst is smooth-walled and, if not fluctuant, more or less elastic in consistency. Not infrequently it is surrounded by chronic mastitis. In all cases of doubt between cyst and solid swelling, or between innocent and malignant cyst, the lump should be excised for microscopical examination.

(1) **Retention Cysts due to duct obstruction.**—Blockage of a large duct near the nipple may give rise (a) to a simple subareolar cyst, or, if lactation be in progress, (b) to a galactocele. Obstruction of small ducts as in chronic mastitis may cause (c) involution cysts; in exaggerated cases the process of fibrosis and cyst-formation advances until (d) polycystic disease of the breast is developed. Retention cysts may also arise in connexion with duct blockage by (e) neoplasms, such as duct papillomata or fibro-adenomata. Chronic encysted abscess, whether due to *B. tuberculosis* or to the staphylococcus, is to be remembered in this connexion.

(a) **SIMPLE SUBAREOLAR CYST.**—A superficial, obviously fluctuant, single cyst of about one inch diameter is easily palpable in the neighbourhood of the areola. The corresponding mammary lobe often shows chronic mastitis. Occasionally a serous exudate can be expressed from the nipple, but this phenomenon is much commoner in cysts due to deep papillomata.

Treatment.—Excision through a radial excision, followed by microscopic confirmation of the diagnosis, is the only satisfactory treatment.

(b) **GALACTOCELE.**—A fluctuant, obviously cystic swelling appears near the nipple during pregnancy or lactation, and may persist long after cessation of suckling. It is painless, rounded and smooth, moves with the nipple, and has any diameter up to about 3 in. A milky discharge can often be expressed from the nipple, and on incision altered milky or buttery contents are found enclosed in a firm, fibrous wall lined with flattened epithelium.

Treatment.—Excision through a radial incision is the most satisfactory treatment. In

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some cases, however, evacuation by aspiration, followed by pressure strapping and inhibition of lactation, is sufficient.

(c) **Inflammatory involution cysts** and (d) **polycystic disease** have already been considered under **Chronic Mastitis** (see p. 186).

(2) **Serous Cysts** occasionally result from the dilatation of lymph spaces; they rarely attain a prominent size, cause symptoms, or require excision.

(3) **Neoplastic Cysts**.—As already mentioned, *retention* cysts may follow duct obstruction by such tumours as **fibro-adenomata** and **duct papillomata**, but cyst-formation may also be caused by *degenerative* processes, colloid or hæmorrhagic, within the substance of a carcinoma or sarcoma. They require treatment appropriate to the causative neoplasm.

(4) **Hydatid Cysts** of the breast are very rare. The cyst is a painless, firm, elastic, round swelling of very slow growth. Until mixed infection has occurred there are no adhesions to skin or pectoral fascia and no axillary adenitis. Fluctuation may be difficult to demonstrate until the cyst has reached a considerable size. Discharge from the nipple is absent throughout. On aspiration a clear watery fluid, free from albumin but often showing hooklets, is withdrawn. (See also **HYDATID DISEASE**.)

Treatment.—The cyst should be excised, whole, through a radial incision before supuration or rupture has occurred. If an abscess be present, it should be opened, thoroughly cleansed, drained, and encouraged to heal from the bottom. If sinuses are present, they should be slit up into their ultimate ramifications; but if the whole breast be riddled it should be removed.

7. INNOCENT TUMOURS

Hard Fibro-adenoma.—This very common neoplasm forms an encapsulated tumour, composed chiefly of firm connective tissue enclosing crushed relics of glandular elements.

Clinical features.—Attention is usually attracted to the lump by chance, or occasionally by neuralgic pain with menstrual exacerbations. The growth begins after puberty, and practically always before the age of 30, but it may remain unnoticed until later. Though occasionally deeply placed and perhaps pushing the breast forwards, a fibro-adenoma is nearly always superficially situated, especially near the nipple, near one margin of the gland

or in its axillary tail. It tends to grow towards the surface until it may eventually present as a visible lobulated tumour. More than one fibro-adenoma may be found, and both breasts may be affected. The tumour is readily palpable with the flat palm; it is smooth, of rounded or lobulated shape, of firm and uniform consistence, and of medium size—it rarely has a maximum diameter of more than 3 in. Except for the frequent presence of chronic mastitis in the corresponding lobe, the surrounding tissues are little affected; the tumour moves freely in the breast, often like a piece of soap slipping from the fingers; it is but loosely connected with breast tissue, and can be moved apart from the nipple; neither skin nor fasciæ are adherent to it, and the axillary glands are not enlarged. On *incision* it is seen to be composed of fibrous laminæ more or less concentrically arranged in dense whorls which enclose small chinks representing imprisoned and choked lumina. The mass is of an opaque, pinkish-white colour in section, and is surrounded by a well-developed capsule from which it can readily be enucleated.

Treatment.—In a young nonchalant patient a fibro-adenoma may be left untouched, though it should be vigilantly watched; it should be excised if first discovered after the age of 30 or if it causes neuralgic pain or mental apprehension. When there is much neighbouring mastitis and the diagnosis between fibro-adenoma and scirrhus is uncertain, the tumour should be removed immediately and examined microscopically. Usually excision is performed through a radial incision directly over the growth; occasionally, when the fibro-adenoma is deep or when a concealed scar is desired, but never when carcinoma is suspected, the surgeon may reach the tumour from behind by incising through the submammary groove and turning up the breast from the chest wall.

Soft Fibro-adenoma (erroneously called "adeno-sarcoma," or, when cystic, "cysto-sarcoma").—This rare tumour grows to a much greater size (up to 7 or 8 in. in diameter) and more rapidly than the common hard fibro-adenoma; moreover, it is commoner in women over 30. Like the ordinary variety, however, it causes no adhesions or axillary adenitis and is non-malignant. Owing to its size it may cause dilatation of superficial veins, and may so enlarge the breast as to simulate diffuse hypertrophy; this condition is, however, practically always bilateral, while soft fibro-adenoma is unilateral. Its relative soft-

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ness may prevent the palpable definition of its limits within the gland.

On section the connective tissue is found to be notably cellular rather than fibrous, but the condition is essentially non-malignant in character. It may be diagnosed from sarcoma by absence of evidences of infiltration of adjacent tissues, by freedom from adhesions, by lack of axillary glandular enlargements, and, in section, by the presence of clefts lined with epithelium.

Treatment.—The whole breast should be removed; if on microscopic examination any reasonable suspicion of sarcomatous change remains, the radical clearance of the axilla and removal of all fasciæ and muscles in relation to the breast should be completed.

Cystic Soft Fibro-adenoma.—Small cysts are not infrequent in soft fibro-adenomata; when these attain such size as to be palpably elastic or fluctuant the term "cystic fibro-adenoma" (*syn.* cystadenoma, adenocoele, sero-cystic sarcoma, cysto-sarcoma, etc.) is applied.

Fungating Fibro-adenoma.—Although when fungation through the skin occurs the tumour is most frequently a sarcoma, a cystic fibro-adenoma may rarely fungate as a congested, readily bleeding mass of blunt "foliaceous" projections.

The treatment for both the cystic and fungating varieties is removal of the breast.

Duct Papilloma (*syn.* Papilliferous Cysts; Intracystic Papilloma; Papillary Cystadenoma).

Clinical features.—The patient, a woman of any age, but commonly between 35 and 45, usually seeks advice because of a sanguineous or sometimes serous discharge from the nipple; in some cases this sign is absent. Pain is rare; when present, it may be caused by sudden duct obstruction and distension. In the early stages no tumour is palpable, perhaps for months or years; later, a small, firm, mobile swelling is felt; often an inflammatory induration of the corresponding lobes is distinguishable before the tumour itself. On section, soft papillomatous masses are seen in one or more distended galactophorous ducts. In the dilatation cysts so formed, fluid may be scanty, but usually it is present in definite amount and is either bloodstained, brown, or clear yellow.

Course.—The nipple discharge and chronic induration, with or without a palpable tumour, may remain apparently unchanged for years. In some cases, however, the cyst enlarges to such an extent as to justify the term "cystic

duct papilloma"; it may then press through the skin and the papillomata may protrude to form a congested, bleeding "fungating duct papilloma"; this fungation may be preceded by infection of the cyst contents and the formation of an abscess.

Sooner or later a duct papilloma tends to undergo carcinomatous changes which may not manifest themselves at once; it is therefore always to be treated as a precancerous condition.

Treatment.—In all cases of *persistent* nipple discharge, accompanied by perhaps some neighbouring mastitis, it is advisable to remove the whole breast, even when no tumour is obvious, and then to examine it microscopically.

Pure fibroma, lipoma, myxoma, and pure adenoma are so rare as to require no description here.

8. MALIGNANT TUMOURS

Carcinoma is very commonly, sarcoma infrequently, and endothelioma rarely seen. All present the usual malignant features; they tend to grow progressively and ultimately to kill the patient; their cells develop along lines foreign or "atypical" to the organ harbouring them, infiltrate adjacent tissues in an un-governed, irregular manner, are guided by lymphatics or blood-vessels to distant viscera, and there cause secondary growths similar to the parent neoplasm.

Carcinoma.—Chronic mastitis, especially the cystic varieties, and duct papilloma are often precancerous conditions, and their presence should lead to anxious search for malignant change. Mammary cancer is commonest near the menopause, but may appear at any age after 20. Rare in men, in women it ranks only second to uterine carcinoma in prevalence. Most frequent in the superior external quadrant, it may appear in mammary tissue anywhere.

Brief morbid anatomy.—Eliminating growths originating in the skin of the nipple or breast, mammary carcinomata are divisible by their structure, intensity of stroma-reaction, and believed origin into the following groups:

i. SPHEROIDAL-CELLED: ARISING IN ACINI—

(a) *Scirrhus*: ramifying columns of spheroidal or flattened, comparatively active cells; but intercolumnar stroma well marked. On section a scirrhus is hard, cuts in a creaking manner, and presents a rather concave, mottled greyish or yellowish-white

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surface from which a thin "cancer-juice" can often be scraped or squeezed.

- (b) *Atrophic Scirrhus*: stroma dense and excessive, cellular elements more or less choked. As a result of the extreme fibrosis the tumour grows very slowly and is very hard, and perhaps nodular; sometimes it forms merely a concave disc. Owing to the fibrous tissue contraction, the whole breast may appear atrophied.
- (c) *Alveolar, Acinous or Adeno-carcinoma*: the cells maintain some alveolar arrangement instead of a grouping in irregular columns.
- (d) *Medullary, Encephaloid or Soft Carcinoma*: the stroma is frail; rapidly growing, loosely coherent cellular elements predominate. On section this variety is soft and perhaps friable; it cuts without creaking.
- (e) *Carcinoma Mastitoides* is a very malignant, rapid, soft carcinoma, probably arising at many points simultaneously (multicentric origin).
- (f) *Colloid Carcinoma*: partly degenerate.

ii. DUCT CARCINOMA, COLUMNAR-CELLED: ARISING IN DUCTS—

- (a) The more characteristic variety of duct cancer arises in the *larger ducts*, after dry cancerous degeneration of duct papillomata. From the edge of a duct cyst packed with papillomata or their degenerate remains, columnar epithelial cells irrupt into the surrounding tissues. Such a carcinoma principally attacks elderly women, grows slowly, affects skin, axillary glands, and nipple late, and has a relatively benign prognosis.
- (b) Carcinoma may also originate in the small ducts; the irregularly ingrowing cells may then be low, cubical, or almost spheroidal, and the neoplasm is differentiated with difficulty from one arising in acini.

Most surgeons agree that duct carcinoma is much rarer than that of acinous origin; but Cheatle believes that many tumours usually classed with the latter really begin in the small ducts.

Clinical features.—These may first be described in terms of a SCIRRHUS OF MODERATE FIRMNESS AND RAPIDITY, the essential differ-

ences and the other types being indicated subsequently.

The *first sign* observed is often a well-developed tumour, discovered by chance. Sometimes, however, an occasional neuralgic or aching pain attracts attention. Unfortunately the disease has usually secured a firm hold before it is detected.

The *tumour* in its early stage, before obvious secondary changes such as adhesions have supervened, may be mistaken for a fibro-adenoma or may be overlooked in a mass of chronic inflammatory tissue. But, with the patient lying on a firm couch, the neoplasm is palpable with the flat hand as a single definitely hard, nodular lump of irregular and often craggy outline with no special tendency to a lobular distribution. In nearly half the cases it is found in the superior external quadrant of the breast, but it may attack any part of the organ. It is attached to and moves with the mamma, but is not, at first, adherent to skin or underlying fascia. Localized swelling may become visible, but when fibrotic changes are extreme, as in atrophic scirrhus, the whole breast appears flattened and diminished in size.

Pain is absent in the earliest stages and may never be prominent. Often, however, it is neuralgic, sometimes referred to shoulder or scapula, and, in the later stages, may be severe, continuous and exhausting.

The *nipple* moves with the tumour. Later its retraction and elevation above the level of its fellow by fibrotic contraction constitute two of the most characteristic signs of cancer.

The *lymphatic glands* are soon involved, long before they become readily palpable. Enlargement of the axillary group, the commonest lymphatic complication, is best detected by relaxing the patient's muscles, pressing the examining finger-tips high into the armpit and gently drawing them down each axillary wall in turn; the glands are felt as small, insensitive hard nodes slipping under the fingers; later they become matted together in a mass, and perhaps adherent to chest-wall and skin. In very late cases the opposite armpit may be affected. The subclavicular glands must always be sought, for they may be soon involved, either directly from the breast or secondarily to the axillary group. Affection of the supraclavicular glands occurs later, but is not an insuperable objection to the radical operation.

Adherence to underlying fascia is a comparatively early and very important sign, determined by attempting to move the tumour

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along the direction of the pectoralis fibres after the muscle has been stretched by holding the arm above the head or by causing the patient to press her palms firmly together.

Adhesion of skin to growth, as shown by the dimpling and puckering elicited by attempts to raise the skin between finger and thumb, occurs relatively early. A coarsely-pitted, orange-rind or pig-skin appearance arises in some cases, probably as the result of interference by perilymphatic fibrosis with lymph drainage of the skin. Occasionally this progresses to a condition of *cancerous pachydermia*, in which the skin, perhaps over a large area, is tough, stiff and leathery, though not necessarily yet itself cancerous; when the cuticle itself is generally infiltrated with carcinoma the term "cancer-en-cuirasse" is applicable. A commoner form of involvement of skin with actual cancer is the cutaneous *nodule*: a localized swelling appears as the result of lymphatic permeation along the suspensory ligaments of Astley Cooper. This becomes more and more raised and bossy, and red or purple, until it finally ulcerates. *Ulceration* occurs late, but is not necessarily a contraindication to radical operation. The ulcer is ragged, irregular, and hard-edged, with sloughy base and offensive watery, often bloodstained discharge.

Cachexia may be absent until very late. It is due to pain, sleeplessness, and secondary infection with pyogenic organisms.

Brawny arm occurs when the growth has already exceeded the bounds of operability. As a result of lymphatic obstruction by perilymphatic fibrosis the arm becomes swollen, brawny, painful, and progressively useless.

Bone-metastases may occur anywhere, but especially in the sternum, ribs, spine, and upper ends of the femur and humerus. They may cause deep aching or neuralgic pain, or may only be detected by radiography when fracture from slight injury has attracted attention. In elderly women "spontaneous" fracture should always lead to search for an undiscovered scirrhus. In the long bones metastases are endosteal in position.

Secondary growths may also develop in *lungs* and *pleura*, in the *liver*, centrally or superficially, or in other abdominal viscera, especially those in the pelvis.

An *ATROPHIC SCIRRHUS* presents many of the signs of an ordinary scirrhus, but is even harder, much slower in growth, and more favourable in prognosis. It causes shrinking and flattening of the breast, but may produce

no serious symptoms until the patient dies of intercurrent disease or until perhaps a "spontaneous" fracture enforces attention.

A *MEDULLARY OR ENCEPHALOID CANCER* is much more serious. It is soft and lobulated, attacks relatively young women, grows and disseminates itself rapidly, and comparatively soon attains a fatal issue.

MULTICENTRIC CARCINOMA (Carcinoma Mastitoides, Mastitis Carcinomatosa) is a rare but hopelessly fatal disease, in which the cancer originates at many points simultaneously, quickly infiltrates the whole breast, and runs its course in a few weeks. It attacks young women, usually in connexion with child-bearing, and may at first be readily confused with acute mastitis. The whole breast is swollen, hot and red, "cancer-en-cuirasse" rapidly develops, and metastases soon cause death.

A *DUCT CARCINOMA* of the more typical columnar-celled large-duct variety is comparatively slow and benign. It frequently supervenes in duct papilloma without any obvious sudden change. Retraction of the nipple is rare, cutaneous and fascial adherence is late, and glandular enlargements are tardy. The commonest, but not invariable, symptom is a bloodstained exudation from the nipple. The tumour may not be palpable for a long time.

The small-duct type of cubical or mixed cubical and spheroidal cells conforms more closely to the scirrhus in clinical characters.

Treatment.—Whatever the clinical or pathological type, the proper treatment in all operable cases is radical removal of the breast in one mass with all underlying muscles, a very wide circle of fascia, all the contents of the axilla and, if necessary, of the supra-clavicular triangle. *No operations that fail to remove all adjacent fascias and muscles and to clear the axilla must be performed.* Several courses of X-ray treatment should follow.

Limits of operability.—A case ceases to be radically operable when—

- (1) General ill-health or pulmonary or cardiac disease is extreme.
- (2) The growth is not only fixed to the pectorals and fascia, but also to several ribs. One or two ribs may be excised with the mass, but their involvement very materially diminishes the prospects of a cure.
- (3) Axillary glands are firmly fixed to the chest-wall.
- (4) Secondary skin nodules exist more

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than 3 in. from the primary growth (Handley).

- (5) Metastases are present in thoracic or abdominal viscera, in spine or in bones.
- (6) There is a large and *fixed* mass of supra-clavicular glands. If these glands are not massive and fixed they may be excised with a moderate prospect of cure.
- (7) The presence of well-established "brawny arm" suggests that permeation with cancer cells has spread so widely as to render cure improbable.

Ulceration of the growth is not in itself a contraindication to operation.

Inoperable cancer.—Radium and X-ray treatment may delay progress, but my experience of these measures has not been encouraging. Oöphorectomy has not established itself as a retarding measure. Attention must therefore be paid to the relief of pain, sleeplessness, sepsis and fœtor, by careful dressing and the use of analgesics; morphia should be reserved as long as possible.

Sarcoma.—Sarcoma shows a predilection for the fourth and fifth decades, but is fortunately uncommon. It may take the form of—

- (a) A very malignant, quickly growing round-celled sarcoma, with early metastases and a tendency to the formation of hæmorrhagic or sometimes alveolar cysts within the tumour.
- (b) A less malignant, slower fibro-sarcoma or spindle-celled sarcoma.
- (c) Very rarely a chondro-sarcoma, containing islands of cartilage, but not, thereby, necessarily less malignant.

Clinical features.—The round-celled variety is the commonest, and may be taken as the type. The breast becomes widely infiltrated and considerably enlarged, and its superficial veins often distended. In it is found a hemispherical, occasionally pulsatile tumour of smooth or lobulated rather than nodular outline, and often of great size. Its consistency may be evenly firm, or softened in places by the presence of cysts. Retraction of the nipple and adherence of the skin are absent. Although the axillary glands are not usually enlarged they are sometimes palpable, and the detection of enlarged glands in this situation does not exclude the diagnosis of sarcoma. In the later stages, fungation through the reddened attenuated skin occurs and a purplish, hæmorrhagic,

sometimes sloughy mass protrudes. Metastases via the blood-stream are common; death is due to cachexia, hæmorrhage, or to metastases in important organs.

The *fibroid variety* is of slower development and may remain for some time as a comparatively small firm tumour, not unlike a fibroadenoma in character. Soon, however, more rapid infiltration occurs and the subsequent course resembles that described above.

Treatment.—In all cases radical removal, as for carcinoma, with clearance of the axilla must be done.

Endothelioma.—This tumour arises in the endothelium lining lymphatics and blood-vessels. Although possibly some tumours hitherto ranked as alveolar carcinomata were really endotheliomata, this disease is rare. The clinical manifestations and treatment are similar to those of carcinomata, but possibly the prognosis is more favourable.

9. DISEASES OF THE NIPPLE

Among **developmental errors** of the nipple may be mentioned: (a) *Undue shortness*. (b) *Umbilication*, in which it lies in a depression surrounded by a moat-like valley. (c) *In-vagination*, in which it is so far buried in the breast as to form a depression instead of an elevation.

Retraction is an acquired condition, and often due to carcinoma, though it may result from scarring, syphilis, or the contraction of cystic remains.

Fissures are discussed under **INFANT FEEDING**, and **chancre** at p. 187.

Papilloma and **Epithelioma** may occur as elsewhere on the skin. They present the usual features, and demand appropriate treatment.

Simple Eczema of the nipple resembles the disease elsewhere in its signs and treatment. (See **ECZEMA**.)

Paget's Disease.—This very intractable condition affects the nipple and surrounding skin, especially in women over 40. It is essentially a malignant phenomenon, for sooner or later in practically every case a carcinoma, usually a scirrhus or a duct cancer, is discovered somewhere in the breast. The scirrhus is often extremely atrophic, which explains the chronicity of the dermatitis and the tardy manifestation of the causative growth.

The affected area has usually a raw appearance, a smooth or finely granular surface, a bright-red colour, and is thickly coated with a clear yellow gummy exudate, which may dry

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to form scabs. Occasionally vesicles like those of eczema, or rarely dry scales resembling those of psoriasis, are seen. The condition is always unilateral; usually it is free from subjective symptoms, though sometimes it causes burning and itching. It is not amenable to the treatment proper for simple eczema.

Treatment.—The whole breast should be removed, with neighbouring muscles and fasciæ and the contents of the axilla. This radical operation should be performed even if the primary cancerous condition is impalpable; less drastic treatment implies unjustifiable risks of later carcinomatous developments.

C. C. CHOYCE.

BRIGHT'S DISEASE (see NEPHRITIS).

BROMIDROSIS (see SWEAT-GLANDS, AFFECTIONS OF).

BROMISM (see DRUG ERUPTIONS).

BRONCHIECTASIS (including Bronchiolectasis).—The dilatation of the bronchi constituting bronchiectasis may be local or universal, and may be cylindrical or saccular in form. Bronchiolectasis is a similar condition in which the smaller branches of the bronchial tree are affected.

Etiology.—The affection occurs at all ages and in both sexes, but is more common in males than in females. A history of a preceding infection, such as pneumonia, bronchopneumonia, or a pleural effusion in which the lung is compressed, is generally obtained. The condition seems to occur more frequently after a broncho-pneumonia which accompanies measles than after that accompanying other diseases. In a few cases no history of preceding disease can be elicited.

A foreign body, such as a tooth or a piece of inhaled bone, is likely to set up ulceration at the point of fixation, and this, if not rapidly fatal, may lead to a bronchiectasis. Similarly, infected material inhaled at the time of an operation—as, for instance, on the antrum of Highmore—is especially likely to produce it.

Pathology.—Originally it was held that bronchiectases were produced by the contraction of fibrous tissue extending between the bronchus and the pleura. Such an explanation depended upon the supposition that the visceral pleura is always adherent to the parietal. This, however, is not the case. The formation of these dilatations is always associated with a cough and some disease which causes inflamma-

tion of the lung tissue. The increased intrapulmonary pressure produced by coughing is sufficient to account for the dilatation of the bronchial tubes in an area in which inflammation produces a destruction of the resisting power of the bronchial wall and consolidation in the air-cells. One or several cavities may be found, the shapes of which are irregular. A "cylindrical" bronchiectasis results when the dilatation is uniform; the enlargement is then spindle-shaped. When the dilatation extends from only one side of the bronchus, a saccular cavity will be formed. The mouth of this may be relatively large or small. Fibrous trabeculae in the walls may produce loculation. The surface of the cavity is smooth. When the cavities are few in number they are frequently of considerable size. Variations occur from this condition to that of a large area converted into a number of small cavities, the "honeycomb" lung. These two forms may be found either in the upper or in the lower lobes, but the latter are pre-eminently the site for bronchiectasis.

The natural constituents of the bronchial wall disappear and are replaced by fibrous tissue, the surface of which is lined by flattened epithelium.

Symptomatology. *First stage.*—With ectasis of one of the larger bronchial tubes there may be no symptoms. In such a case the cavity is dry and no secretion is present. The usual complaint is that whenever a cold develops it is rapidly followed by a cough with a good deal of expectoration. This is at first purulent and later becomes more mucoid in type. In the course of a few weeks it gradually diminishes and may again disappear. In the *second stage* there is constantly a certain amount of mucoid expectoration, which at times, usually after a cold, becomes purulent. It is more difficult to get rid of this purulent character than in the former stage, but after some months the expectoration may disappear almost entirely. In the *third stage* of the disease purulent expectoration is constantly present, and in the most advanced cases is copious and presents characters which are typical. When allowed to stand it settles in three layers, the top layer being frothy, the middle one an opalescent fluid, while at the bottom of the jar the material is of a greenish colour and consists largely of solid particles. The expectoration occurs in a very characteristic manner. Following forced inspiration and expiration, such as are incidental to exertion, singing, or laughing, and also alteration in the position of the axis of the

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trunk, as in bending down or reclining, a sensation of something being present in the throat is experienced and a quantity of material is expectorated. The explanation is that the cavity containing the secretion is lined by a mucous membrane which is insensitive. When, however, the contents are expelled from the cavity, they impinge upon healthy mucous membrane which is normally sensitive to foreign material, and coughing is produced. After the cavity has been emptied the cough is diminished for the time being.

When the dilatations are limited to the smaller tubes this characteristic method of expelling the contents does not prevail. All the cavities are never properly emptied simultaneously, and the cough and expectoration of purulent fluid are more continuous. In both forms the cough may disturb sleep. It is severe in the morning as a result of the nocturnal accumulation.

Beyond the cough and the troublesome expectoration there may be no symptoms. The patient may be well nourished and able to do a fair amount of work. As a rule, there is a slight degree of breathlessness and of anæmia with its associated symptoms. No rise of temperature at night occurs, sweating is not complained of, and the general functions of the body are normal. When the infection spreads to the adjacent lung, producing a form of broncho-pneumonia, the symptoms of this condition will be observed, but they will be less prominent than in the case of an otherwise healthy individual, probably because the body has become accustomed to the presence of this permanent infection. When a large amount of suppuration is constantly present, lardaceous changes may set in. Reviewed as a whole, it is extraordinary how slight are the general symptoms caused by the disease, even when it is considerable in extent.

Diagnosis.—Bronchiectasis affecting a large tube and one near the centre of the lung may be quite undetectable, and if quiescent may not even be suspected. Single cavities which are active will give rise to expectoration typical in its substance and method of evacuation as described above. When a cavity is empty, all the characteristic signs of a vomica may be detected; when it is full, these signs are absent. Such signs do not distinguish between a bronchiectasis and a cavity of another nature. Although bronchiectases are usually found in the lower part of the lower lobes, and other cavities, especially those due to tubercle, at the apices

of the lobes, the ultimate diagnosis rests upon the examination of the sputum. The absence of tubercle bacilli and the presence of a mixed flora is strongly suggestive of bronchiectasis. The general condition of the patient with bronchiectasis is good, whereas the signs, if due to tubercle, would indicate an advanced case.

When the smaller tubes are affected and "honeycomb" lung results, it is usually impossible for the cavities to be emptied in the circumstances described. In such cases the sputum is often nummular and generally contains only one type of organism, the cough is more persistent, and the lesion is generally found at the base of the lung, either posteriorly or in the neighbourhood of the axilla. Very similar signs may be found in the case of *resolving broncho-pneumonia*, but a re-examination at the end of a week or so will reveal the true condition. An *empyema opening into the lung* suddenly causes the evacuation of a large quantity of purulent sputum; in the case of bronchiectasis the quantity increases on successive days and has not such an obvious appearance of pus. In a ruptured empyema there is also a tendency for the expectoration to recur at longer intervals (two or three days); in bronchiectasis it is a daily occurrence. An *hydatid cyst* is recognized by the character of the sputum, which contains hooklets or parts of the cyst-wall. The sputum of *actinomycosis* is also characteristic. *Malignant growths in the lung* produce bronchiectasis. In such an instance the wasting is rapid and the general condition correspondingly worse.

Pus may accumulate in a recess in the respiratory area other than the lung, such as in a supratonsillar pocket. In this event confusion may arise through a similar method of expectoration, and a diagnosis of a small bronchiectasis be made. Repeated careful examinations of the throat will obviate the error.

On the whole, a patient with considerable expectoration and cough, exhibiting a large number of signs but in good general condition, is most likely to be the subject of bronchiectasis. If a tuberculous infection be superimposed the bacilli may only be found with difficulty.

A warning must here be given against exploring the chest in cases of bronchiectasis; there is considerable liability to the development of cerebral abscess when it is attempted.

Prognosis.—This is a difficult matter

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When the cavity is single and not of large size the duration of life is not greatly shortened. Whenever a cavity is actively secreting pus the infection may spread to the adjacent lung and set up broncho-pneumonia; it is remarkable, however, what resistance is shown by patients having a chronic infection of the lung. Hæmorrhage sufficient to be fatal is uncommon. There is considerable liability to the development of metastatic abscess, especially in the brain. It should not be forgotten that lardaceous diseases or tuberculosis may supervene.

The heart may become dilated—a tendency which is roughly proportionate to the amount of lung destroyed and to the dyspnoea resulting from this destruction. Death by failure of the right side of the heart is a common method of termination.

Treatment.—Treatment is directed towards obviating activity in the cavities, and relieving the condition when the cavities become active. The greatest precaution should be taken to prevent the patient from acquiring any infection, however mild it may be, from those about him. He should therefore be removed from the vicinity of any who show signs of developing colds, influenza, etc.—a matter of difficulty in practice. The general health should be maintained to the utmost; an open-air life is well tolerated when dyspnoea is not obvious. All foci of sepsis, such as may be found in the gums, tonsils, nasal sinuses, etc., should invariably be searched for and, if present, eradicated. There are no special indications as regards diet.

It is most important that the positions in which the cavities tend to empty themselves should be ascertained, and adopted many times a day by the patient, in order to promote evacuation of the purulent material. The success attending these efforts is considerable when the cavities are few and large, but disappointing in bronchiolectasis. When the cavities become active during the winter months, additional treatment should be adopted. For an hour, night and morning, the patient should wear a Burney Yeo open inhaler to the sponge of which are applied a few drops of the following inhalation, viz.: creosote 3 dr., thymol 3 dr., carbolic acid 1 dr., spirit of chloroform to 1 oz. The liquid is best applied to the sponge a few drops at a time with the aid of a match or wooden penholder. If necessary, the Burney Yeo inhaler may be worn for a much longer period, or even continuously. It is

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important that it should be made of perforated zinc; and until the patient is accustomed to place the proper quantity of the liquid on the sponge, the face should be rubbed with vaseline over the area on which the edges of the mask impinge, otherwise the skin may be burned. From the end of September to the end of April the patient should take a minim of creosote in a drachm of cod-liver oil thrice daily after food. On the least sign of any infection, such as a cold or a pyrexial attack, the patient should be ordered to bed and kept there until the practitioner is satisfied that all danger is over. He should be conservative in allowing the patient to get up. After an acute exacerbation tonics are of value, and for this purpose large doses of the tincture of perchloride of iron up to 30 min. three times a day, for a period of a week, are beneficial.

Vaccines have a considerable effect on the general condition of the patient. Unless, however, evacuation by the means indicated can be attained, their use will be disappointing if it is anticipated that they will immediately reduce cough and expectoration. They are, however, well worth trying. The antiseptic inhalations usually diminish the variety of organisms present. When the sputum shows that the varieties of organisms are constant, it is advisable to take stock vaccines prepared from a virulent strain obtained from a similar infection, instead of employing an autogenous vaccine.

Should broncho-pneumonia, hæmorrhage, etc., arise in these cases, the treatment appropriate to the complication should be applied, without, as far as possible, relaxing the treatment indicated above.

Operative measures have been successful in a few cases, but the risk attending them is considerable, and they should therefore not be undertaken lightly.

CHARLTON BRISCOE.

BRONCHITIS.—Inflammation of the bronchial mucous membrane may be acute or chronic; in chronic bronchitis the expectoration may be so excessive as to lead to bronchorrhœa. When the sputum has an offensive odour the term "fœtid" bronchitis is employed.

1. ACUTE BRONCHITIS

Etiology.—Acute bronchitis is commonly preceded by a sore throat and in a mild degree is a concomitant of an ordinary cold, extension taking place through the larynx and trachea.

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It is also liable to develop in the course of any acute illness, e.g. typhoid fever, and is especially common with measles and whooping-cough.

Pathology.—The changes found in the mucous membranes are—hyperæmia in the first stage, congestion and infiltration by leucocytes and desquamation of the ciliated epithelium in the second stage, and finally resolution. With resolution the inflammatory changes disappear and regeneration of the lining membrane takes place. As a rule the attack clears up, but it may become chronic, and exacerbations are then liable to occur. Tubes of any size may be affected, and, should bronchitis of smaller tubes be present, bronchopneumonia may supervene. When bronchitis recurs frequently, the submucous connective tissue is increased and the epithelium tends to become thinner and less stratified, and to be replaced by non-ciliated cells. A permanent state of congestion remains which resembles that seen towards the end of an acute attack.

The pneumococcus is usually found either alone or associated with other organisms. Micrococci catarrhalis, influenza bacilli, streptococci, staphylococci may be found alone, but are more generally associated with pneumococci. The organism of diphtheria is sometimes detected. In this case membrane will be formed, fibrinous in the large tubes, but more purulent if the smaller bronchi are affected.

Symptomatology.—The onset may be either insidious or acute. When developing in the course of another infection it is insidious, but when occurring as a primary condition it may be so acute as to be heralded by a rigor or by vomiting. At the onset pains in the back and limbs are frequent, and throughout the course of the disease there may be pain on swallowing due to laryngitis or to enlarged glands in the mediastinum pressing on the œsophagus. Fever of moderate degree is generally present, and may persist for a few days or several weeks. Complaints are made of dyspnœa which may almost amount to asthma, of discomfort referred to the upper part of the sternum or between the shoulder-blades, of a general sense of tightness of the chest, and of the air producing a harsh sensation in its upper part. During the first stage, when the mucous membrane is inflamed and congested, there is an irritating non-productive cough owing to the secretion of viscid mucus. In the next stage the sputum becomes less viscid, and yellowish from the addition of leuco-

cytes and serum, and hence the cough is less irritating and more productive. At the end of four or five days the sputum becomes yellow or greenish, more liquid, and easily expectorated; leucocytes and serum are present in greater quantities. As resolution occurs and inflammation of the bronchial tubes decreases, the sputum loses its purulent character, remains thin, then becomes mucoid, is easily expectorated, and gradually lessens. In a mild attack the pulse-rate is not increased, but when the bronchitis is severe it may rise considerably. Respirations are accelerated roughly in proportion to the degree of obstruction. Sleeplessness is common during the first few days, largely owing to the cough. The condition is often associated with nasal catarrh, laryngitis, and tracheitis. The amount of expectoration may not be at all in proportion to the efforts of coughing. In some cases expectoration is small in amount and difficult to bring up; in others it is copious and easily evacuated. Factor is due to the development of putrefactive micro-organisms, the products of which are accountable for the odour; it is frequently not appreciated by the sufferer. The cough is always severe after a night's rest, and in the middle of the night often disturbs sleep. Sudden paroxysms of dyspnœa may develop which are only relieved when the offending material has been expectorated.

Diagnosis.—As a rule, this presents no difficulty, for the symptoms are characteristic. The physical signs are liable to be very in definite, but some rhonchi and sibili are usually audible within forty-eight hours of the onset. Generally, at least the bases of the lungs are the seat of râles or crepitations during some part of the attack. Confusion may arise between cases of acute bronchitis and those of *pressure on the bronchus*, but the history of the case, or the discovery of a cause for bronchial pressure, should speedily solve the difficulty. Dyspnœa may be so great at the onset as to lead to the suspicion that an attack of *asthma* is under observation. It is difficult to separate the two, but at this stage it is not important to make a certain distinction. At the onset many cases of bronchitis are associated with spasm, and, on the other hand, an attack of asthma no less frequently precedes bronchitis. It should be remembered that a weak condition induced by *wasting diseases* may be complicated by bronchitis, and that bronchitis may be the predominant symptom of such infections as *typhoid fever* and *measles*. Should a

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weakly person seek treatment for bronchitis, the primary cause should be sought.

Prognosis.—The ordinary case of acute bronchitis occurring in a healthy subject is almost sure to end in recovery. In the aged, in young children, or in weakly adults the prognosis may be serious. The disease is dangerous when it begins in the finest tubes or by spreading involves them, for bronchopneumonia is to be feared. The association of other diseases, such as measles, whooping-cough, etc., tends to increase the gravity of the affection. Bronchitis is very likely to be the determining cause of death in such conditions as emphysema, diabetes, chronic nephritis, and cirrhosis of the liver; the prognosis should then be guarded, even though the pulmonary condition is apparently mild.

Treatment.—In all cases it is advisable that the patient be put to bed. The room should be airy and capable of ventilation, and its temperature kept at 60°–65° F. The bed should be so placed that it is neither in a direct draught nor in a corner where the air does not circulate. A purge should be given immediately, followed by a saline draught in six or eight hours. It is of the greatest comfort to the patient that the air be warm and moist. If the atmosphere does not conform to these conditions, a fire should be lighted and a kettle with a spout projecting into the room allowed to simmer, so that steam may be given off. When the bed is placed in a tent, as may be done in the case of children, the kettle should not be too close to it, otherwise the child may burn its hand. Too much moisture in the air is depressing and harmful; the kettle should therefore not be kept in position continuously, but should only be in operation for periods of three-quarters of an hour to an hour, separated by similar periods. A poultice applied over the upper part of the sternum and between the shoulder-blades affords considerable relief. It should be graduated with the strength of the individual, and when placed over the sternum should be light. The first of the poultices may contain a little mustard. They should be changed every two hours. Failing poultices, a mustard-leaf or a hot fomentation on which a little turpentine is sprinkled may be applied; to prevent excoriation this should be removed five or ten minutes after severe discomfort is complained of. Woollen jackets are advisable. In preparing them, allowance should be made for the fact that they may have to be changed

at short intervals if much perspiration should occur. The patient should be allowed to assume whatever posture is most comfortable.

Considerable relief will follow the use of an inhaler in which hot water is placed, the vapour being inhaled. Many varieties of inhaler are obtainable. Of more importance than the kind of appliance is the instruction that, while inhaling, the patient should breathe quietly. Inhalation is of the greatest value where the larynx and trachea are involved. Friar's balsam, some of the volatile oils, or even turpentine may be added in the proportion of one teaspoonful to a pint of hot water. The water should be at a temperature of 140° F.

Although the diet at this stage should be very limited, it is of great importance that a large quantity of liquid, such as hot lemonade or barley-water, should be imbibed. As alkalis are of considerable assistance, bicarbonate of soda may be added in a proportion of 20 gr. to half a pint. Four or five pints of lemonade may easily be consumed by an average adult during the twenty-four hours. It is important to obtain rest at the very beginning of the attack when the cough is unproductive and there are few signs in the lungs. Dover's powder 15 gr., or morphia and ipecacuanha lozenges, may be ordered; the latter have the advantage that the patient may be given one every quarter of an hour until the desired result is obtained. Aspirin may be prescribed, or a hot bath given, prior to taking to bed, if severe pains are complained of in the body and limbs.

Many drugs have been recommended for this condition. If the patient is a strong young adult, small doses (15 min.) of tartar emetic may be given, and repeated every two hours for six doses. In the case of weak patients, tartar emetic is best avoided. Alkalis and stimulants are the most useful drugs at this stage. It is advisable that the expectorants ordered at the onset be given every hour or two hours when the patient is awake, until expectoration becomes easy; they should only be discontinued should they produce dyspnoea. A useful prescription such as the following may form a basis of the drug treatment:—

R \bar{y} Liq. ammon. acet. ℥i.
Sod. citrat. gr. x.
Sod. bicarb. gr. x.
Sp. ammon. aromat. ℥x.
Syr. tolut. ℥x.
Aq. ad ℥ss.

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This should be taken in a wineglassful of water every hour for twenty-four hours, and subsequently at four-hourly intervals. Three minims of ipecacuanha wine is a useful addition; it may produce nausea. Tincture of belladonna or tincture of stramonium (5 min.) may be included if there is much spasm. These drugs should be omitted if they cause depression, vomiting, or toxic symptoms. Spiritus etheris or spiritus vini rectificatus may be added if stimulation is necessary. Hypodermic injections of strychnine ($\frac{1}{2}$ gr.) or ether (10 min.) are useful when depression or collapse is present. In the case of elderly persons it is important to see that nourishment is taken. This should consist of broths, beef tea, warm milk diluted, lightly poached eggs, Benger's or Allenburys' food, or some of the dried preparations made from milk. Stimulants may be required, and the usual quantity taken should not be cancelled. It should be insisted upon that during some part of the day the patient should sit up in bed or be propped on pillows. Whenever possible, for elderly persons and children two nurses should be engaged.

This régime may be continued until the breathing is relieved and the expectoration becomes easier, when the diet may be increased by the addition of fish, poultry, fruit, toast, milk puddings, and so forth. The expectorant mixture may be given less frequently, and the ipecacuanha, belladonna, or stramonium discontinued. It should be insisted on that the patient expectorates into a suitable receptacle the material coughed up, which should on no account be swallowed.

Towards the end of the seventh or tenth day, when the expectoration is becoming watery and the temperature has fallen to normal, the diet may be further increased. The expectorant mixture may be superseded by tonics containing strychnine and arsenic and, for those under 50 years of age, iron. Provided there are no digestive symptoms, large doses of iron (tincture ferri perchloridi 20 min.) will often produce a marked improvement. The tincture should not be continued for more than a week at this strength. Stimulants in the form of spirits or port wine are also useful. For the young especially, cod-liver oil in doses of 1-2 dr. has a considerable value. Before the patient settles down for the night it is advisable to give him a dose of the original medicine and to make him expire deeply, thus emptying the bronchial tubes of any secretion they may contain. A glass of hot lemonade at this time

will cause expectoration occurring during the night to be more readily coughed up. On waking, a large cup of tea will have a similar effect; the patient should, as far as possible, remain quiet for a period of half an hour after taking it.

In the course of four or five days to a week after the cough and expectoration have ceased the patient may be allowed to get up in the bedroom, and two or three days later may resort to a neighbouring room. Here a fire should be lighted three or four hours before the patient's entry. During the next day or two, according to circumstances, a descent to a room on the ground floor may be made under similar precautions, and thereafter advance made by easy stages until the patient goes out and resumes his normal life. For most patients over the age of infancy a visit to the seaside is advisable. Exception must be made in the case of subjects over 50 years of age and of those with a tendency to shortness of breath or with cardiac complications; such patients will benefit more by a change inland. Discretion must also be used in choosing the place to which the patient is sent, the West or the South Coast being preferable to the East during the winter months.

A common and annoying sequela is a persistent hard, dry cough, especially on waking and after food. This is generally relieved by an alkaline nasal douche and the elimination of carbohydrates temporarily from the diet.

Should the cough and expectoration continue, benefit may be obtained by the employment of vaccines. This will entail an examination of the sputum and possibly a culture to detect the organisms, which at this stage are usually the pneumococcus and the micrococcus catarrhalis. When a patient is liable to develop recurrent attacks, precautionary measures should be taken. Alternatives are the administration of a minim of creosote in a drachm of cod-liver oil thrice daily from the middle of October to the middle of April, and the prophylactic injection of a vaccine at intervals throughout the winter. The most useful vaccine is one containing pneumococci, micrococcus catarrhalis, and the bacillus of influenza. When it is known that the patient is prone to infection by another organism such as the streptococcus, this should be added to the vaccine. Injections should be made every three to five weeks. Either of these lines of treatment, if continued for two or three years, will not infrequently overcome the tendency to

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bronchitis. Before the adoption of either of these methods, a thorough examination should be made to exclude the presence of sepsis in the respiratory tract, the naso-pharynx, tonsils, or cranial sinuses. The presence of pyorrhoea either in the patient himself or in one of the members of his household should be excluded.

2. CHRONIC BRONCHITIS

Etiology.—Chronic bronchitis may occur either as a number of recurrent attacks or as a prolonged attack with only slight remissions. Frequently some other condition is present, such as emphysema, bronchial dilatation, morbus cordis, chronic infection of the respiratory tract, or persistent ill-health associated with tuberculosis, malignant disease, nephritis, cirrhosis of the liver, etc. In some cases the bronchitis is associated with recurrent attacks of bronchial spasm.

Pathology.—The mucous membrane of the bronchial tubes is reddened, congested, and covered with exudate, which may be mucoid or semi-purulent. The inflammation tends to spread to the finer parts of the bronchial system. The submucous connective tissue is increased, blood-vessels appear to be more numerous, the muscle and cartilage of the bronchial wall may be absorbed, and the ciliated epithelium lining the tube may be replaced by cubical epithelium. The mucous membrane contains fewer layers of cells than normal. The pneumococcus is the prevailing organism in the sputum, but the micrococcus catarrhalis, streptococci, and less well-known bacteria may be present. To some of the latter is due the offensive smell which occasionally accompanies the affection.

Symptomatology.—Frequent cough and expectoration are the characteristic symptoms, and, except for the distress caused by the cough and some shortness of breath, the patient's general condition is often satisfactory. Occasionally there are night-sweats and dyspepsia. The latter is probably due to the swallowing of infected mucus, with subsequent subacute infection of the stomach.

Diagnosis.—This presents little difficulty. *Bronchiectasis* and *tuberculosis* have to be distinguished from it. *Bronchiectasis* can, as a rule, be diagnosed by the characteristic method of expectoration. In all cases of chronic bronchitis a thorough examination of the sputum should be made, in view of the possible association of tuberculosis. Many cases diagnosed as chronic bronchitis are in reality

cases of *chronic pharyngitis*. It must always be remembered that some other pathological state may be undermining the patient's health and rendering him liable to prolonged infection.

Prognosis.—Chronic bronchitis occurring in youth may cease when adult life is reached, and never recur; but those who have been affected early in life are more prone to develop the condition afresh after the age of 40. At the other extreme of life, chronic bronchitis is not of very serious import provided that there is no underlying lesion, for most elderly people suffer from it to some degree. It may, however, be the terminal illness which carries off the patient, generally by the supervention of broncho-pneumonia. The prognosis in middle life is not serious unless emphysema, which causes a greater liability to heart failure, is added. A patient who suffers from chronic bronchitis may be troubled considerably by the frequent cough and shortness of breath, especially if the bronchitis be associated with spasm. Any affection, such as an ordinary cold, may have an adverse influence, and short attacks of broncho-pneumonia may ensue.

Treatment.—Whenever possible, the patient should be sent abroad during the cold months of the year. A dry climate, such as that of Egypt, should be selected for those whose expectoration is profuse. If cough is the troublesome symptom, a moister climate such as that of the Riviera, Algiers, or the Canary Islands may be selected. In England the south and west coasts afford the most suitable conditions. For those who must remain at work and are unable to choose a locality, steps must be taken to guard against infection, and, if possible, so to arrange that, should any cold develop, attention to business may be dispensed with. The patient must take every care to avoid getting wet; he should pay special attention to his boots, which should be well soled and oiled, and should keep an extra pair of boots and socks at his place of occupation and carry a serviceable overcoat or mackintosh. A rigorous search should be made for any focus of infection; this, if found, should be eradicated, and any underlying conditions such as nephritis treated. Fresh air is not less important than in tuberculosis, and, where dyspnoea is not great, should be insisted upon. The diet needs little restriction, but articles which cause flatulence or indigestion must be excluded; it is especially necessary to limit the amount of starchy food. A little stimulant is beneficial. Direct treatment for the arrest of

BRUISES

the disease consists in the use of tonics, cod-liver oil, to which creosote may be added, and inhalations similar to those employed for bronchiectasis (q.v.). Considerable benefit accrues from the administration of potassium iodide when any slight exacerbation occurs. Vaccine treatment is useful, but should be employed rather as a prophylactic than curatively. Attention should be paid to the bowels. When exacerbations occur they should be treated in the manner recommended for acute bronchitis. Various antiseptics, other than creosote, have been recommended, such as garlic, oil of allyl, and cubebs; they are worth trying.

CHARLTON BRISCOE.

BRONCHOOELE (see GOITRE).

BRONHO - PNEUMONIA (see under PNEUMONIA).

BRONZED DIABETES (see HÆMOCHROMATOSIS).

BROWN-SÉQUARD SYNDROME (see SPINAL CORD, LOCAL LESIONS OF).

BRUISES.—Contusion is the result of a blunt force applied to the tissues, which compresses or stretches them beyond their powers of resistance. It is especially liable to occur where soft tissues overlie bone. The skin is more elastic than subjacent tissues, and in most cases escapes injury. The blood- and lymph-capillaries give way, the intercellular tissue is lacerated, and effusion of blood and lymph takes place. Admixture with lymph renders blood less coagulable, and consequently the bleeding continues. The exudation may spread far from the point of injury, travelling by gravity along lymph-spaces; for example, discoloration of the eyelids often follows a blow on the vertex. Very extensive bruising occurs in hæmophilia as the result of trivial injuries. The symptoms of a bruise are pain and a feeling of tension in the part. The colour becomes purple, then in a day or so changes through bluish-black and green to yellow. Staining may remain for a considerable time. The function of the part is interfered with.

Treatment.—In the early stages pressure will limit the exudation. It is best applied by firm bandaging over a thick layer of wool. The edges of the wool should not be covered with the bandage, especially in the case of the limbs, for the hard margin of the bandage may cause venous obstruction and increase the effusion.

BUBO

Cold obtained by an ice-bag or cold water may limit exudation, but pressure is more effective. Glycerin and belladonna (ext. belladonnæ viridis $\frac{1}{2}$ oz., water 1 dr., glycerin to 1 oz.) may relieve pain, but should not be applied to the eye. Later on, the disappearance of the discoloration is assisted by massage.

C. A. PANNETT.

BRUISES FROM THE MEDICO-LEGAL STANDPOINT (see INJURIES FROM THE MEDICO-LEGAL STANDPOINT).

BUBO.—A suppurative inflammation of the glands in the groin. The glands are arranged in two groups, one parallel with Poupart's ligament, draining the lymph from the abdominal wall below the level of the umbilicus, the buttocks, anal region, perineum, and genitals; the other, vertically disposed, draining lymph from the lower limb. A septic focus in any of these regions may provide a source from which pyogenic organisms may proceed to the glands. Gonorrhœa, soft sore, a septic focus in the lower limb, and affections of the anus are the common causes.

Symptoms.—Pain in the groin, stiffness, and inability to extend the hip completely are accompanied by a hard, tender swelling in the groin. When the infective process breaks through the glandular capsule to the periglandular tissues the swelling, which at first was rounded, becomes more diffuse and ill-defined. The skin becomes œdematous and red, and fluctuation appears. The temperature is raised.

Diagnosis.—A suppurating gland situated over the crural canal may be difficult to distinguish from a strangulated femoral hernia, especially when omentum alone is in the sac. The finding of the focus of infection, and the absence of abdominal pain and signs of intestinal obstruction, usually suffice to make the diagnosis clear.

Treatment.—The causative condition must be treated. Rest in bed is essential. Boric-acid fomentations should be applied to the part. Incision frequently becomes necessary; it should be made vertically over the most prominent part of the swelling, and a drainage-tube should be inserted for forty-eight hours. Excision of the glands is not to be recommended, and might prove a difficult dissection.

C. A. PANNETT.

BÜHL'S DISEASE (see JAUNDICE).

BULBAR PARALYSIS

BULBAR PARALYSIS is usually a part of progressive muscular atrophy, and is due to the same pathological lesions. The wasting of the bulbar muscles sometimes precedes the affections of the muscles of the limbs. (See **MUSCULAR ATROPHY, PROGRESSIVE**.)

BULIMIA (see **STOMACH, FUNCTIONAL DISORDERS OF**).

BUNIONS (see **HALLUX VALGUS**).

BUPHTHALMOS (see **GLAUCOMA**).

BURNS AND SCALDS.—Scalds are due to the application of moist heat (e.g. boiling water, steam). Burns are produced by the action of dry heat, as in radiation from the sun or from a fire; or by contact with actual flames, heated solid objects, or the burning gas resulting from an explosion; or by friction, lightning, electricity, or X-rays.

Pathology.—Four degrees of burns are described. In the *first degree* erythema alone occurs; in the *second* there is vesication; in the *third* a slough is produced of varying depth of tissue; and in the *fourth* the whole of a portion of the body, such as a limb, is charred (carbonization). Resulting from the destruction of cells and solution of red corpuscles by the high temperature, certain toxic substances pass into the circulation, producing lesions of the internal organs. These include congestion and ecchymosis of the mucous and serous surfaces, hyperæmia of the meninges and cerebral oedema, cloudy swelling of the kidneys and liver, enlargement of the lymphatic glands, and, very rarely, duodenal ulcer. A rise in the specific gravity of the plasma occurs.

Symptomatology.—In burns of the *first degree* there is erythema of the affected area, which becomes swollen and feels tense. Pain may be acute for a few hours; it is made worse by the application of warmth. The local condition subsides in a few days, when desquamation occurs. There is no scarring. In burns of the *second degree* vesicles and bullæ appear on the erythematous area. In some of them the exuded lymph clots to a jelly. The pain is severe, increased by warmth, and may last three or four days. The blisters dry up, forming a crust which separates in about a week. There is no scarring unless infection occurs, leading to the formation of a septic ulcer. In burns of the *third degree* the devitalized area is insensative, shrunken, and whitish-yellow or yellowish-black in colour. Around

BURNS AND SCALDS

the eschar burns of the first and second degrees are seen. The slough gradually separates, to the accompaniment of a mild infective process, and a granulating wound is left. Erosion of blood-vessels, secondary hæmorrhage or thrombosis may result. A scar always remains, and if the slough be deep or extensive severe deformities may follow. The neck may be drawn down to the trunk, or an arm fixed to the side; contractures of joints and ectropion are common, and cheloid is apt to develop in the scars. Scalds of the pharynx and larynx from drinking hot liquids are serious because of the danger of oedema of the glottis supervening and causing death.

The constitutional symptoms are proportional to the severity of the injury. Shock may be very pronounced. The temperature is subnormal at first, but after recovery from the shock it is raised by toxæmia and may remain high for some days. Thirst is complained of. There may be hæmoglobinuria and albumosuria, due to solution of the red corpuscles and disintegration of the slough. In fatal cases there are apathy, delirium passing into coma, clonic convulsions, diarrhoea, and vomiting, the last symptom being a valuable indication of the seriousness of the case.

Prognosis.—Death occurs *always* if more than one-half the surface of the body be burned, *usually* if half be implicated, *very often* if the burn affects one-third. It may result from shock, toxæmia, fall of blood-pressure, or sepsis. Recovery may be attained only with the formation of ugly or crippling deformities, and healing of large ulcers may not be possible unless some plastic operation be done.

Treatment.—It is of prime importance to combat **shock**. This can be done by administering as much fluid to the patient as he will take. Saline per rectum is best, but he should be allowed to drink as much as he will, and recourse to subcutaneous or intravenous infusion may be necessary. Fluid must in some manner be introduced. Pain should be controlled by morphia. For children of less than one year $\frac{1}{1000}$ gr. morphia tartrate per month of life may be given hypodermically, or tincture of opium by the mouth ($\frac{1}{2}$ min. up to 6 months, 1 min. from 6 to 12 months). For combating shock in children a hot-air bath is useful provided that morphia has first been given. A cradle is placed over the child, and one or more in-

candescent electric lamps swung from it under the bedclothes.

Local treatment differs according to the severity of the injury. In burns of the *first degree* an ointment is indicated, either boric-acid (one-fourth of the B.P. strength) or eucalyptus ointment. In burns of the *second degree* infection must be prevented, or scarring will result. Hence it is proper to give an anæsthetic and clean up thoroughly the surrounding skin with ether soap and carbolic lotion (1 in 20). In hairy regions shaving must be done. Blisters should be punctured near their margins, but the separated cuticle should be left. After thorough purification, either gauze soaked in picric-acid solution (picric acid 20 gr., absolute alcohol 6 dr., water to 10 oz.) is applied, or a dressing of dry sterilized gauze thickly sprinkled with equal parts of starch and bismuth subcarbonate powder. Either dressing should be left untouched for a week, or longer, unless the appearance of much discharge necessitates its removal. In burns of the *third degree*, under anæsthesia the injured area and its surroundings should be thoroughly purified and dressed either with cyanide or picric-acid gauze. The dressing need not be changed until the granulations begin to secrete somewhat profusely, as they will do after some days. After this, frequent dry dressings are necessary. When the wounds have taken on the characteristics of the healing ulcer, epithelialization may be stimulated by the application of scarlet-red ointment (amido-azotoluol 8 per cent. in vaselin). Weak boric-acid ointment (one-fourth the B.P. strength) should be applied all round the wound to within $\frac{1}{4}$ in. from its margin. The red ointment is then applied on a piece of lint cut to the shape of the ulcer, but overlapping its margin for $\frac{1}{4}$ in. all round. Every seventh day boric-acid ointment alone should be used. The paraffin treatment of burns received an extensive trial during the late War, and the success following its use was so great that the method is to be strongly advocated. Under anæsthesia, if necessary, the burn and its neighbourhood should be rendered surgically clean. Flavine (1 in 1,000) is recommended as an antiseptic. The following paraffin wax, melted and at a temperature of 56°-60° F., should be applied :

Beta-naphthol	.	.	.	0.25	per cent.
Eucalyptus oil	.	.	.	2.0	" "
Olive oil	.	.	.	5.0	" "
Paraffinum molle	.	.	.	25.0	" "
Paraffinum durum	.	.	.	67.75	" "

A brush may be used for the application, or the melted wax may be sprayed from a container kept warm by a water jacket. A thin layer of wool is now applied to the burn, then another painting with wax is done, and the whole is covered by a thick layer of wool. The dressing is renewed every twenty-four hours. As it will not adhere, its removal will not be painful if the first layer of wool is sufficiently thick. In cases of long duration, epithelialization may be stimulated by washing the ulcer with 1-per-cent. scarlet-red at each dressing before applying the wax. Skin-grafting may be necessary, and is often advisable. Sometimes large plastic operations will have to be performed to correct or conceal deformities.

In scalds of the mouth and pharynx the contingency of tracheotomy being suddenly called for must be borne in mind.

C. A. PANNETT.

BURNS FROM THE MEDICO-LEGAL STANDPOINT.—Lesions produced by the application of heat or chemical substances to the surface of the body are in law referred to as burns, whether they are caused by dry or by moist heat (*see* previous article).

Identification of the agent.—On a body exposed to the action of fire any degree of burning may be found, from simple erythema to complete charring or carbonization of the tissues. The hair and clothing in these cases will probably also show the effects of fire.

Scalding fluids and steam produce more or less widespread vesication, the clothing is not burned, and there are no deposits of carbonaceous material on the surface of the body. Where vesication is the only sign present it is important to remember that it may be produced by friction, or by exposure of a portion of the body to the action of decomposing fluids, e.g. in bedridden patients and children who have been allowed to lie in their own discharges.

The lesions produced by corrosives will depend on the particular substance employed. Vesication is rarely found when corrosive mineral acids or alkalis have been used, but may occur when Greek fire (phosphorus dissolved in carbon disulphide) has been employed. When the carbon disulphide evaporates, the deposited phosphorus is oxidized by the air and bursts into flame, so that burning by actual fire results. The colour of the stains on the skin and clothing will assist in determining the

nature of the corrosives used. On the skin, sulphuric acid produces a dark or even black colour, hydrochloric or carbolic acid a white or greyish-white, and nitric acid a yellow. On cloth, sulphuric acid produces a dirty-brown stain with red edges, hydrochloric acid a red stain which becomes brown in time, and nitric acid a yellow, orange-red, or brown coloration. The colour produced on cloth is apt to be varied by the original colour of the fabric.

Fatal cases.—Death may occur from shock, and children and the aged succumb more easily than robust adults. Even superficial burns are dangerous to life if a large portion of the surface of the body is involved. If the patient survive the immediate effects, death may still result from toxæmia, sepsis, exhaustion, secondary hæmorrhage, inflammation of serous membranes, or perforating ulcer of the duodenum.

Were the lesions produced before or after death?—If the patient dies it will be necessary to decide whether the lesions found were produced before or after death. Burns produced during life can usually be recognized by the presence of signs of vital reaction—swelling, hyperæmia, etc. When the burning has produced only a superficial redness the coloration is apt to disappear with the onset of hypostasis. If vesication has resulted the blisters have an inflammatory areola, contain serous fluid in which albumin and chlorides are found, and present an injected base. The application of heat to a dead body may cause a blister, but there will be no inflammatory areola and the blister will contain only air or a small amount of non-albuminous fluid free from chlorides. The presence of suppuration indicates that life has been prolonged for some time after the burning.

Post-mortem examination.—The presence of burns on a body does not necessarily indicate, of course, that burning was the cause of death. In burning buildings death may result from asphyxia from the gases (CO and CO₂) of combustion, or from pressure on the chest while a crowd is attempting to escape, or from injuries produced by falling down and being trampled on, or from injuries caused by falling masonry, or from syncope, etc. In cases of murder by mechanical violence or poisoning, an attempt may be made to burn the body so as to destroy the evidence of the true cause of death. For these reasons a post-mortem examination should be made in every case.

That death was due to burning will be determined by the character and extent of the lesions, the signs that the burns were produced during life, and the absence of signs of other cause of death.

The chief external lesions have already been referred to. Care should be taken to distinguish between solution of continuity of tissue the result of fire, and wounds produced by mechanical violence. If the skin and subcutaneous tissues have been burst open by the effect of heat, the sides of the wounds will be irregular and will often be connected by bridges of the more resistant tissues, e.g. tendons, etc.; they will thus be distinguished from wounds made by a cutting instrument. Fractures of bones may be caused by falling down, or by falling beams, etc., or by heat, and their nature is difficult to determine when there has been much charring of the body. The bodies of those who have been burned to death often exhibit "heat stiffening," the limbs being fixed in a position of contortion, and a "pugilistic" attitude is sometimes assumed.

The internal organs are congested, especially the brain, lungs, and kidneys. The blood is coagulated and often brighter than usual from the destruction of the red corpuscles by the high temperature; and this bright coloration occurs independently of the presence of CO-hæmoglobin.

Accident, suicide, or homicide?—No definite rules can be formulated for determining whether burns are accidental, suicidal, or homicidal. Lay evidence is likely to be more helpful than medical in determining the point. The largest number of cases are accidental, homicidal cases are not uncommon, suicidal cases occur but rarely.

A. ALLISON.

BURSAL ENLARGEMENTS.—This condition may result from (1) contusion, (2) acute bursitis, (3) chronic bursitis.

(1) A *contusion* is seen most often in the olecranon and prepatellar bursæ following a fall. There is a sudden swelling of the sac, which becomes filled with blood (bursal hæmatoma). Absorption of the blood is often incomplete, and thickening of the wall results from organization of the lining clot. Suppuration may occur, especially when there is an abrasion of the overlying skin. Treatment consists in elastic pressure (see BRUISE) and the application of cold. Later, warmth and massage hasten absorption. (2) *Acute*

BURSAL ENLARGEMENTS

bursitis is due to infection, directly from a wound, or by extension from a neighbouring septic focus, or is blood-borne (rheumatism, gonorrhoea, syphilis). It is accompanied by an acute serous effusion in blood and mild pyogenic infections, and in gout. In the more severe pyogenic infections acute suppurative bursitis occurs. The wall is converted into granulation tissue, and the bursa is filled with pus, which breaks through into the surrounding tissue, giving rise to a cellulitis. (3) *Chronic bursitis* is due to repeated small injuries, and is seen in the well-known examples of housemaid's knee (prepatellar bursitis), miner's elbow (olecranon bursitis), and weaver's bottom (ischial bursitis). The effusion is straw-coloured. The bursal wall is thickened and fibrous, sometimes calcified, and villi often project from it. Adventitious bursae are sometimes formed over bony points subjected to pressure. They are liable to become inflamed. The best-known example of this is a bursa which forms over the head of the metatarsal bone in hallux valgus. A tuberculous bursitis occurs, and this is associated usually with neighbouring bone or joint disease; syphilis may cause a gummatous thickening. These two forms are not further considered here.

Symptomatology.—The swelling corresponds to the shape and position of the bursa. The prepatellar bursa lies in front of the lower half of the patella, the olecranon bursa over the point of the elbow, and the ischial bursa forms a swelling over the tuber ischii. When the subacromial bursa is enlarged there is a fullness under the deltoid, and the swelling projects from under the anterior margin of the muscle. Abduction is limited, but rotation with the arm to the side is free. The ilio-psoas bursa forms a swelling in the upper and outer part of Scarpa's triangle. Flexion and internal rotation of the hip-joint are free, the other movements restricted. The semimembranosus bursa projects backwards from the popliteal space, close to the outer side of the tendon of the muscle. It is tense on extension of the knee, and perhaps can be reduced into the joint on flexion.

In *acute* bursitis the swelling appears rapidly. There is tenderness, and the overlying skin is reddened. Signs of the causal disease (gonorrhoea, rheumatism) may be found. When suppuration occurs the temperature is high, the pain becomes worse and the skin is cede-

matous. In the *chronic* form the enlargement is slow, and there is no pain.

Treatment.—In *acute* bursitis rest is essential, a back-splint being employed for prepatellar bursitis, a sling for olecranon bursitis. The arm should be bandaged to the side when the subacromial bursa is affected. For bursitis of the psoas bursa rest in bed is necessary. Aspiration, followed by elastic pressure by wool and a bandage, is good treatment. Any underlying disease should be dealt with. When suppuration occurs, incisions should be made, but care must be taken not to open a bursa unnecessarily (see ABSCESS, PREPATELLAR).

In *chronic* bursitis also rest should be given as described above, and irritation of the bursa, e.g. kneeling when the prepatellar bursa is affected, prohibited. The application of strapping over Scott's ointment applied on lint to the swelling promotes absorption, and is particularly useful in conjunction with aspiration. Counter-irritation by painting with tincture of iodine combined with pressure is less effective. When the swelling persists in spite of treatment, removal is recommended.

The prepatellar and the olecranon bursa can be exposed by turning down a flap outlined by an incision convex upwards. The bursa should be removed intact. The operation is a simple one. Removal of the semimembranosus bursa is a more difficult dissection, and should only be done when every precaution can be taken against infection, because the knee-joint frequently communicates with the bursa. The subacromial bursa is reached by an incision through the anterior fibres of the deltoid. If this bursa requires removal it should be remembered that the underlying cause is sometimes an injury of the infraspinatus tendon, which may require a suture. Excision of the psoas bursa is seldom required. Access is gained by a vertical incision in the upper part of the thigh between the femoral artery on the inner side, and the anterior crural nerve on the outer side. As the injection of irritants into the sac is painful and unreliable, it has been abandoned in the treatment of chronic bursitis. When the bursa associated with hallux valgus is acutely inflamed it is treated like any other bursa until the acute process has subsided, when further operative measures may be considered. It is useless to remove this bursa unless the deformity is corrected at the same time (see HALLUX VALGUS).

C. A. PARNELL.

CAISSON DISEASE

CAISSON DISEASE (*syn.* Diver's Palsy).—Workers in compressed air are liable on, or shortly after, decompression to various symptoms. Compression, or the raising of the air-pressure in the caisson, or diving-suit, is attended with no risk to the workmen, and may be carried out at the rate of increase of one or two atmospheres per minute. The most unpleasant symptom is caused by pressure on the membrana tympani, which may rupture if the Eustachian tube be not patent enough to permit the pressure in the middle ear promptly to become equal to that outside. Divers are sometimes exposed to a pressure of five or six atmospheres (25 fathoms of sea-water); in caissons the pressure rarely reaches more than three atmospheres.

During compression the tissues become saturated through the lungs with the gases in the air. During decompression the reverse takes place, the excess of gas is liberated and escapes by the blood-stream and lungs. If it escapes too quickly from the tissues it forms bubbles in the blood and tissue-fluids and various symptoms follow. Obviously the risk varies with the degree of saturation and the rapidity of decompression. It also depends on the activity of the circulation—the more active the less risk; and as some tissues—e.g. fat—absorb nitrogen with peculiar readiness, risk is greater for the corpulent. No risk at all attaches to rapid decompression from a pressure of two atmospheres (absolute).

Dyspnoea and death may occur from extensive air embolism in the pulmonary capillaries. The commonest complaint is of pains in the limbs and joints, known as "bends," which are attributed to bubbles in the synovial fluids. The chief and most enduring symptoms are caused by bubbles forming emboli in the capillaries in the brain and spinal cord. Headache, vertigo, vomiting, and collapse sometimes occur, and motor and sensory paralysis due to lesions in the brain or spinal cord may develop.

Prognosis as to recovery depends on the severity of the symptoms. Often complete recovery occurs in a few days, even when the symptoms have been very severe. Once symptoms have become established, the clinical picture is that of focal nervous disease.

The only specific treatment is recompression. This should be carried out immediately symptoms appear, and should be followed by

a more gradual decompression. If this is not feasible, large doses of ergot are recommended.

FRANK C. PURSER.

OALABAR SWELLINGS (*see* FILARIASIS).

OALCINOSIS.—A very rare disease, the essential feature of which is calcification of the subcutaneous tissues. It is met with in children as well as in adults. Small, hard, shotty, tender nodules are felt beneath the skin. They have often a linear arrangement, but may be scattered indiscriminately over the trunk and limbs. The nodules may be preceded by a purplish-red discoloration and a sclerodermatous condition of the skin, and some fixation of the joints has been noted. At first movable, the nodules later become fixed; the skin over them may inflame and ulcerate, and a short sinus result, discharging pus and chalk. Little is known as to the future of these cases. In most of those described, the disease has progressed slowly, but improvement has been recorded. No special treatment has been found of value.

FREDERICK LANGMEAD.

OALOULI (*see* GALL-STONES; PANCREATIC CALCULI; SALIVARY CALCULI; URINARY CALCULI).

CALMETTE'S OPHTHALMIC REACTION (*see* SEROLOGICAL DIAGNOSIS).

OAMMIDGE'S REACTION (*see* URINE, EXAMINATION OF).

CANCER (*see* under individual organs).

CANORUM ORIS (*see* STOMATITIS AND GLOSSITIS).

CANTHARIDES POISONING (*see* POISONS AND POISONING).

CAPILLARY BRONCHITIS (*see* Lobular Pneumonia, under PNEUMONIA).

OAPUT SUCCEDANEUM (*see* BIRTH INJURIES).

CARBOLIO ACID POISONING (*see* POISONS AND POISONING).

CARBON DIOXIDE SNOW, TREATMENT BY.—This form of treatment is suitable for many types of skin disease. The necessary apparatus consists of (1) a cylinder of CO₂, (2) a chamois leather bag, and (3) several small boxwood moulds, square or round, the latter

CARBON DIOXIDE SNOW

about the circumference of a shilling. The bag is attached to the cylinder by tape, and the carbonic acid projected into it, where it forms a flaky deposit. The CO_2 must leave the cylinder in liquid form, otherwise no snow will be obtained: for this reason some cylinders should be placed in a horizontal, others in a vertical, position. The snow is scraped from the bag with a spoon and pounded into a solid stick in the mould selected. The resulting cylinder, conveniently about 1 in. long, is wrapped in cotton wool and held between the finger and thumb, and so applied to the skin. The action is twofold, destructive and "alterative." The former result, suitable in rodent ulcer, is obtained by a firm pressure lasting 1-2 minutes; it should not be attempted without some experience. The latter effect, desirable in the treatment of *nævi*, lupus erythematosus, etc., is obtained by light pressure for 5-40 seconds. As a result of the operation the skin first becomes white and hard, then swells, and finally may form a blister. The after-treatment consists of opening the blister if necessary. Scarring may be minimized by antiseptic precautions. Protection with a dry gauze dressing is required where a deep reaction has taken place.

A large lesion must be treated bit by bit; the same place can be re-treated after an interval of three weeks or when completely healed. The common "strawberry mark" always does well under this treatment; an exposure of 20 sec. is suitable. Subcutaneous *nævi* and "port-wine marks" are less satisfactory. In lupus erythematosus an application of about 20 sec. is given, and the results are excellent. An application for 5-20 sec. gives good results in chloasma. In the treatment of large warts the snow may be applied for 60 sec., if confined strictly to the lesion. Carcinoma, like warty *nævi* and hairy moles, demands deep pressure of one minute's duration, always cautiously employed.

If the treatment is followed by sepsis, ulcers and unsightly scars result, but under ordinary conditions the procedure is perfectly safe.

H. MACCORMAC.

CARBON MONOXIDE POISONING
(see POISONS AND POISONING).

CARBONIC ACID POISONING (see
POISONS AND POISONING).

CARBUNOLE (see FURUNCULOSIS).

CARRIERS OF INFECTION

CARCINOMA (see under the individual organs).

CARDIOLYSIS (see PERICARDITIS).

CARDIO-RESPIRATORY MURMURS
(see HEART, FUNCTIONAL MURMURS OF).

CARIES, DENTAL (see DENTAL CARIES).

CARIES, SPINAL (see SPINAL CARIES).

CARREL-DAKIN TREATMENT (see
WOUNDS, TREATMENT OF).

CARRIERS OF INFECTION.—The terms "germ carriers," "carriers of infection," or simply "carriers" (Fr., *porteurs de germes*; Germ., *Bazillenträger*) designate those who harbour and disseminate the germs of a given disease without presenting any clinical symptoms.

Carriers have been grouped in three classes, as follows:—

1. Those convalescent from the infectious disease in question.
2. Those in the incubation period ("precocious carriers").
3. Those who have not recently had the disease and who do not subsequently develop it.

The existence of the healthy carriers who form the third class has been contested on the grounds that such persons are really suffering from an abortive attack or from some unhealthy local condition which favours the persistence of the germs, such as chronic tonsillitis or rhino-pharyngitis in the case of diphtheria and epidemic cerebro-spinal-meningitis carriers.

The importance of carriers has been well established in the propagation of typhoid fever, diphtheria, cerebro-spinal meningitis, cholera, and dysentery, and it is highly probable that they play a considerable part in the dissemination of other infections, especially of acute poliomyelitis.

The probability is that carriers rarely spread infection by contamination of their hands, hair, clothing, or other external means. The infective nidus in carriers is practically always internal, being situated in the fauces and nasopharynx in diphtheria, cerebro-spinal meningitis and acute poliomyelitis, in the intestine in typhoid, paratyphoid, cholera and dysentery, in the gall-bladder in typhoid and paratyphoid, and in the urinary tract in typhoid. The virus of each disease thus finds a more favourable soil than it would in an extra-

CARRIERS OF INFECTION

corporeal existence, and the latency of its growth and dissemination renders it all the more dangerous.

There can be no doubt that many outbreaks of infectious diseases are due to the unsuspected presence of a carrier.

The number of carriers in a community varies with the prevalence of the infectious disease, being low in non-epidemic times and rising with the accumulation of acute cases. The highest percentage of healthy carriers is likely to be found among the nurses and doctors in attendance.

The duration of infectivity of carriers varies considerably. For this reason they have sometimes been grouped as temporary or chronic, according as their infectivity ceases within or lasts longer than three months.

In diphtheria the bacilli do not, as a rule, persist for long in the throat after subsidence of the acute symptoms, but their disappearance is retarded by local causes, such as large tonsils and adenoids, lesions in the nasal fossae, or dental caries; in such cases the bacilli may be found for months or as long as a year or more.

In typhoid fever, owing to the excellent culture medium afforded by the gall-bladder and urinary passages, cases are on record in which the duration of infectivity has been as long as twenty years; on the other hand, in cholera it is found that the patients rarely continue to excrete bacilli longer than two or three months, or in dysentery more than four or five weeks.

It is important to realize, however, in estimating the duration of infectivity of carriers, that intermittence in the discharge of the infective agent frequently occurs, periods during which cultures of the excretions are negative being followed by positive periods, and vice versa.

Treatment.—The treatment of carriers is eminently unsatisfactory, and many of the so-called "successes" may be attributed either to spontaneous cure or to negative cultures having been obtained during a period of intermission.

In the case of diphtheria occurring in schools or other aggregations of children, carriers should be isolated until at least two negative cultures have been obtained. Meanwhile, local treatment may be employed, such as antiseptic sprays, bacteriocidal lozenges, or the use of living cultures of *Staphylococcus pyogenes aureus*.

CATALEPSY

Enlarged tonsils and adenoids should be removed. Vaccine-therapy has been used both with diphtheria and with typhoid carriers, but the results are inconclusive.

Prolonged isolation for an indefinite period cannot always be enforced, especially in the case of adults who have to earn their living, and in such cases carriers should follow certain rules of conduct. Those in whom the infective nidus is in the intestine or urinary tract, as in typhoid, cholera, or dysentery, should carefully wash their hands after defecation and micturition. As far as possible they should not be allowed to manipulate food intended for others, and therefore should not be engaged as cooks or housemaids. Those in whom the infective nidus is in the throat should refrain from kissing and from the use in common of eating and drinking vessels and pipes.

Animals as well as men may act as carriers of infection. Most of the examples of this kind are to be found in tropical or at least exotic medicine, such as the goat in undulant fever, anopheles in malaria, stegomyia in yellow fever, fleas in plague, and lice in typhus and recurrent fever.

In this country the most important animal carrier is the house-fly, which is often responsible for epidemics of typhoid fever and infantile summer diarrhoea. J. D. ROLLESTON.

CARUNOLE, URETHRAL (see URETHRAL CARUNCLE).

CATALEPSY.—A condition in which the limbs can be passively moved without apparent resistance into any desired position, this position being subsequently preserved for an indefinite period. If, for example, the physician raises the patient's arm, it will remain suspended in the air until another position is similarly impressed upon it. The length of time during which such a position will be maintained, if there is no further external interference, is very variable, but it may extend to hours. Owing to the fact that the patient reacts to every movement impressed upon him by the physician in a way precisely comparable to the plasticity of a waxen figure, the name *cerea flexibilitas* (waxy flexibility) is applied to this phenomenon.

Catalepsy is met with in several distinct conditions. It can be produced artificially in hypnosis, and is indeed generally present when any degree of hypnosis beyond the lightest has

CATARACT

been induced. Probably here it is mainly a product of suggestion. Catalepsy also occurs in hysteria, where it may be a feature of trance-like states, or of the so-called ecstatic attitudes sometimes observed. It is found most frequently, however, in the catatonic variety of dementia præcox, and constitutes a common symptom in the stuporose phases of that disorder (*see* CATATONIA).

BERNARD HART.

CATAPHORESIS (*see* IONIZATION).

CATARACT.—An opacity of the crystalline lens; the term is not properly applied to opacities situated on the surface of the lens but outside the capsule.

Symptomatology and diagnosis.—Small stationary opacities may give rise to no defect of sight, but in progressive cataracts, of which the senile form is a typical example, the patient complains of a gradual deterioration of vision, sometimes associated with spots in the visual field. Such spots, in contradistinction to those which result from vitreous opacities, do not continue to float after the eyes have come to rest.

Small or incipient cataracts are most easily detected by illuminating the dilated pupil with light from the ophthalmoscope mirror held at a distance of 2 or 3 ft.; the opacities show up dark against the red background, and may then be examined more closely by the direct method of ophthalmoscopy (*see* EYE, EXAMINATION OF); for faint opacities the plane mirror is to be preferred.

To ascertain the position of the opacity the light of a lamp situated in front, and to one side of the patient's face, should be concentrated on the eye by means of a convex lens (oblique illumination); if the observer have good binocular vision he will have no difficulty in judging whether the opacity is superficially or deeply situated in the lens; by this method also an opacity in the cornea or anterior chamber is readily distinguished from a true cataract. With the ophthalmoscope also the position of an opacity can be estimated by the observation of its parallactic movement with reference to the plane of the pupil. It is obvious that if, while the illuminated pupil is kept under observation, the patient moves his eye upwards (or the observer his eye downwards), an opacity in front of the plane of the iris (e.g. in the cornea) will appear to move upwards, while an opacity behind that level

will appear to move downwards; and, moreover, the greater the distance of the opacity from the pupillary plane the greater will be the apparent movement. Again, an opacity which continues to float after the eye has come to rest cannot be in the solid lens, but must be in one of the fluid humours, almost certainly the vitreous.

With the gradual maturation of the cataract a white reflex appears in the pupil. Even in the absence of opacity, however, there is frequently a strong reflex from a sclerosed senile lens; in this case the use of the ophthalmoscope will show a normal red reflex. A diagnosis of cataract should never, therefore, be made till this test has been applied. A cataract is mature when the opacity reaches quite up to the anterior capsule; at this stage also the anterior chamber, previously somewhat shallow owing to swelling of the lens, becomes of more normal depth.

Varieties.—Since the fundamental pathology of cataract is obscure, and similar forms may result from different causes, the most serviceable classification is based upon the capsular, cortical, or central situation of the opacity.

1. **Capsular cataract** results from a proliferation of the epithelial cells which line the anterior capsule. It is found especially after perforation of corneal ulcers and in hypermature cataracts, and takes the form of small chalky-white spots and areas in the most superficial layers of the lens. It occurs with especial frequency at the anterior pole (*anterior polar cataract*).

2. **Cortical cataracts.**—Of these the most important is *senile cataract*, which results from a breaking down of the cortical lens fibres, commencing in the deeper layers, and most frequently at the equator; sometimes, however, in the antero-posterior axis behind or in front of the nucleus. In the former case, with the ophthalmoscope, spokes of opacity project into the illuminated pupil from the periphery; in the latter a more diffuse granular opacity occupies the centre of the pupil. Axial opacities naturally cause earlier and greater deterioration of vision. *Diabetic cataract* is of the same type.

Secondary or complicated cataract results from malnutrition of the lens when other disease—irido-cyclitis, detachment of the retina, choroido-retinitis, retinitis pigmentosa, etc.—is present in the eye. It commences as a granular or feathery opacity in the posterior cortex, and

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subsequently becomes total. Needless to say, this form of cataract is not amenable to operative treatment.

3. Central and pericentral cataracts.—The prototype of this class is *lamellar or zonular cataract*. It consists of a shell of opacity situated at some depth in the lens, and enclosing a central clearer, but not wholly transparent, area. Outlying opacities (riders) frequently project from its periphery into the clear cortex. It occurs in children, and often dates from an attack of rickets, with convulsions or tetany, in early life. Defects in the enamel of the teeth are frequent, an association to be explained by the common development of the lens and enamel organ from epiblast.

Among rarer types, **congenital cataract** takes a variety of forms—total, central, axial, punctate, etc. **Posterior polar cataract** is due to the persistence of a small portion of the mesoblast which accompanies the foetal hyaloid artery. **Traumatic cataract** is the term applied to the opacity and swelling which follow a wound of the lens.

Treatment.—Congenital stationary cataracts may require no treatment if vision is fairly good ($\frac{1}{8}$ or better). In weighing the question of operation the subsequent loss of accommodation and the necessity of using strong glasses should be set in the balance against a possible improvement in visual acuity. In the case of sharply localized small central opacities, use may be made of the clear periphery of the lens by means of a small optical iridectomy; and on the same principle operation may sometimes be deferred in axial senile cataract by keeping the pupil dilated with atropine. In general, however, the treatment of cataract involves the removal of the opaque lens, which may be carried out—(1) By making repeated small incisions in the capsule with a needle, so that at each operation a small portion of lens substance is brought into contact with the aqueous, and is absorbed. This operation (*discission*) is suitable for the soft lenses of young children. (2) By performing an extensive needling, and a few days later evacuating the swollen lens substance by means of a grooved spoon (*discission with curette evacuation*). Discission is feasible until a "nucleus" has been formed by the gradual physiological sclerosis of the central portion of the lens (about the age of 30), after which it becomes necessary to remove the nucleus and cortex through a large incision at the upper sclero-corneal margin (*extraction*).

It is important that the practitioner should be able to form some judgment as to the suitability of a given case for operation. Juvenile cataracts should be operated on as early as possible, otherwise the benefits of the early use of central vision are lost, and nystagmus and amblyopia may result. In the case of senile cataract the patient should not only perceive the movement of large objects between his eye and the light, but should also be able to point to the direction from which a light is reflected on to the eye (good projection). The pupil should respond with normal briskness to light; an inactive pupil points to imperfect light perception. The pupillary margin, iris, and cornea should be carefully searched for signs of old iritis (posterior synechiae, atrophy, discoloration, deposits of cells or pigment on the back of the cornea). The ocular tension should be tested; cataract in a soft eye is usually secondary and should not be touched; in a glaucomatous eye it may not be the most important factor in the case. The condition of the conjunctiva and lachrymal sac should be investigated; conjunctivitis and lachrymal obstruction must be thoroughly treated before the idea of operation is entertained. The other eye must be tested and examined; the discovery of important fundus changes which are likely to be bilateral (albuminuric or diabetic retinitis, etc.) would preclude operation; if in the second eye cataract is absent or only incipient, and if vision is good, no operation on the first should be advised. The operative prognosis is less favourable in immature than in fully mature cataracts; yet even in the case of immature cataracts operation may be recommended when vision in the other eye has sunk considerably (below $\frac{1}{8}$ or $\frac{1}{4}$) and the patient is debarred from following his employment. Questions of general health must receive consideration; advanced age is scarcely in itself a contra-indication, but the operation, with all its concomitants, may impose too great a strain on the decrepit. Operation should never be undertaken without an examination of the urine for albumin and sugar. Coma and death have not infrequently followed extraction in subjects of diabetes.

GEORGE COATS.

CATARRH, ACUTE SUFFOCATIVE (see SUFFOCATIVE CATARRH, ACUTE).

CATARRHAL FEVER (see CORYZA).

CATARRHAL JAUNDICE (see JAUNDICE).

CATATONIA

CATATONIA.—This term is used in two distinct though closely related senses. In the first place it denotes a symptom-complex comprising a number of characteristic motor phenomena; in the second place it denotes a variety of dementia præcox in which these same motor phenomena are a prominent feature of the symptomatology.

Catatonia as a symptom-complex is characterized by the appearance of a stuporose condition presenting several or all of the following signs: Stereotyped attitudes, the patient preserving for an indefinite period some fixed posture; stereotyped actions, consisting in the constant repetition of some movement, or the monotonous reiteration of some phrase (verbi-geration); negativism, the patient actively resisting whatever is required of him, and often doing its exact opposite—e.g. tightly closing his eyes when asked to open them. Negativism is occasionally varied by the manifestation of certain phenomena to which the term "automatic obedience" has been applied. These are echolalia, in which the patient, instead of answering a question, merely repeats it; echopraxis, in which he immediately reproduces any action which is carried out before him; and *cerea flexibilitas* (catalepsy), in which his limbs preserve for an indefinite period any posture in which they are placed.

This group of symptoms mainly appears in the variety of dementia præcox to which the name of catatonia is also applied, but it is sometimes observed in other disorders, particularly in the confusional psychoses.

Catatonia in its second sense denotes the variety of dementia præcox in whose symptomatology catatonic signs play a prominent part. It is a disorder generally appearing in adolescence or early adult life, and tends to run a course ending most commonly in a terminal dementia, although the prognosis is somewhat less unfavourable in this than in other varieties of dementia præcox (see DEMENTIA PRÆCOX).

BERNARD HART.

CATHETERIZATION.—Catheters are made of rubber, of woven silk covered with varnish (gum-elastic), or of silver.

Sterilization of catheters.—Rubber and silver catheters are sterilized by boiling. Many gum-elastic catheters will not endure boiling, and are best sterilized by formalin vapour. They should be washed and thoroughly dried, and then placed in a long wide glass tube in the stopper of which is a perforated container

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for holding powdered paraform. Sterilization is complete in twenty-four hours. A more rapid formalin method is to have a metal box with a receptacle at one end in which is placed a tablet of paraform that can be vaporized by a spirit lamp. The hot vapour will sterilize in one hour. Before the catheter is used the irritating formalin should be removed by rinsing in sterile water or saline solution. The white French gum-elastic catheters are made of a material which will stand boiling. During this process they should be kept by gauze from coming into contact with each other or the sides of the sterilizer, and only their ends should be grasped by a forceps for removal, or the softened hot varnish will become rough. All catheters immediately after use should be washed thoroughly with soap and water, and their interiors cleansed by syringing through them first water and then some antiseptic solution. Gum-elastic catheters are best preserved dry, lightly dusted with lycopodium powder.

The best lubricant for catheters is a water-soluble one. Sterile oil is a good lubricating agent, but it renders the catheters difficult to cleanse. Either sterile glycerin or, better still, the following lubricant may be used:—

Sterilized glycerin 20 parts.
Tragacanth 2 parts.
Mercuric oxycyanide $\frac{1}{2}$ part.
Sterilized distilled water 100 parts.

Such a lubricant is conveniently put up in sterilized collapsible tubes by the manufacturing chemist.

Passage of the catheter.—Before the catheter is passed the hands must be washed as for an operation. The prepuce is retracted and the meatus and glans are cleansed with mercuric biniodide solution (1 in 1,000). The catheter is lubricated (but not with the fingers); a drop of the lubricant is placed on the meatus. A rubber catheter is grasped by the fingers of the right hand 3 or 4 in. from the eye so that the portion of the catheter which will ultimately lie behind the compressor urethræ muscle may not have come into contact with the hand of the operator. The left hand holds up the penis and exerts slight traction on it. The catheter bit by bit is pushed through the urethra into the bladder. No part of a gum-elastic catheter, owing to its greater rigidity, need be touched except the distal extremity.

It is rather more difficult to pass a metal

instrument. The operator stands to the right of the patient. He holds the distal extremity of the catheter with the shaft horizontal, and parallel with Poupart's ligament on the right side. The beak looks downwards. In this position the point is inserted and the instrument pushed onwards, the penis being dragged up along the shaft. When the beak reaches the bulb the distal extremity is moved so that the shaft comes to lie parallel with the middle line of the body. The shaft is now gradually raised from the horizontal position to the vertical and beyond this, gentle pressure of the point against the face of the compressor urethræ being kept up. Aided perhaps by the fingers of the left hand applied to the perineum, the catheter point will slip through the compressor muscle and glide easily into the bladder.

Choice of catheter.—A metal instrument smaller than No. 8 English (15 French) should never be used except by an expert, and exceedingly seldom by him. A rubber catheter is easily sterilized, and should be used when possible, but it may not possess sufficient rigidity to overcome spasm of the compressor urethræ, for which a gum-elastic catheter is necessary.

For prostatic cases the most useful instrument is a coudé gum-elastic catheter (16 or 18 French). But a rubber catheter often passes quite easily, even when there is retention (10 or 12 E., 16 or 18 F.). The old-fashioned silver prostatic catheter with the large curve should very seldom be used; it will not adapt itself to the sinuous course of the prostatic urethra, and so leads to hæmorrhage. Its passage is also a painful procedure. In the retention of gonorrhœal prostatitis a soft instrument (10 E. or 16 F.) should be passed with the utmost gentleness after washing out the anterior urethra.

In retention from stricture, gum-elastic instruments are employed, beginning with No. 16 F. and passing downwards until filiform bougies are reached. A metal catheter or bougie of smaller size than 8 E. should never be used in these cases.

Method of tying in a catheter.—Rubber or gum-elastic instruments are usually selected when a catheter is to be tied in. Seldom is a metal catheter so used. A rubber catheter is better tolerated by the urethra than one of gum-elastic, because the varnish of the latter soon becomes rough, but a rubber catheter is apt to slip out; a Lucy's stilette will, however, prevent this. Gum-elastic catheters should not be left in longer than four days, and at the end

of this time a new one should be substituted. When tying in a catheter, by trial and error the degree of insertion is determined, and should be such that the eye lies about half an inch beyond the internal vesical sphincter. A piece of narrow tape 8 in. long is then tied round the catheter about half an inch distal to the meatus, but not so firmly as to compress the lumen of the catheter. The two ends, which are left of equal length, are brought along the side of the penis and fixed to it by a piece of strapping about $\frac{1}{2}$ in. wide applied circularly around it just behind the corona glandis.

A silver catheter is tied in by a slightly different method. Two long pieces of tape are taken, one for each side. Each tape is inserted through one of the eyes of the distal end of the catheter and tied to this at its middle. The two ends are then carried up together (the catheter having been inserted to the correct distance) to the side of the root of the scrotum, just where the adductor longus tendon originates. Here they are knotted together. The two ends are carried obliquely round the thigh a little below the fold of the groin and knotted together again over the great trochanter. They are then carried vertically upwards and tied to a tape or bandage going round the waist.

C. A. PANNETT.

CAUDA EQUINA, LESIONS OF (see SPINAL CORD, LOCAL LESIONS OF).

CELLULITIS.—A diffuse inflammation of areolar connective tissue can occur as a cellulitis of the subcutaneous tissue, the orbit, mesentery or retroperitoneal tissue. By the unqualified term cellulitis, however, is usually meant inflammation primarily of the subcutaneous tissue, and only this variety will be considered here.

Etiology.—The affection is usually due to invasion of the subcutaneous tissue by the *Streptococcus pyogenes*, either alone or with staphylococci; rarely by the staphylococcus alone, which is more apt to give rise to a localized abscess. The organisms gain entrance through a wound of the skin, which may be a small abrasion or a prick. An insect bite or sting may provide a path. Occasionally no breach of the surface can be found, and in such cases it is conjectured that the microbes pass through sebaceous glands.

Symptoms.—Pain is felt at the focus of infection, and there rapidly forms a red, tender, brawny area into which serous exudation

CELLULITIS

occurs. The hyperæmic zone is not sharply delimited, and tends to spread. Running from it towards the nearest lymphatic glands are red streaks—the lymphatic vessels, which are inflamed by the passage of the infecting organisms. The glands themselves are enlarged, hard, and tender. The general symptoms vary much in intensity. In the severer cases there is a chill followed by a rise of temperature, which continues irregularly high with remissions while the disease lasts. The process may be arrested at any stage, but, unless treated, shows a great proclivity to spread widely. Both the deep fascia and the skin present a certain resistance to the infective changes, which tends to limit the spread to the subcutaneous tissues. If the barrier of deep fascia be broken through, the inflammatory condition invades the areolar connective-tissue partitions separating the underlying muscles. Necrosis of the overlying skin occurs if the supplying vessels become thrombosed. Here and there in the hot, brawny, tender area foci of softening occur, due to localized collections of pus, and there is a degree of purulent infiltration throughout. Large sloughs of connective tissue may be present under still living but inflamed skin. The degree of extension depends upon the resistance of the patient to the particular infection, and the virulence of the invading organism. Diabetes, chronic nephritis, lack of sufficient food, and specific fevers are very potent in diminishing the powers of resistance, and must be taken into consideration in estimating the probable course of the disease. Extension to deep-seated organs may be very serious, especially in the neck (see LUDWIG'S ANGINA). Thrombosis of the subcutaneous veins may be followed by dislodgment of infected clot and the super-vention of pyæmia.

Cellulitis of the *scalp* deserves special mention. If the infection is limited to the tissues superficial to the epicranial aponeurosis, extension of the process is not necessarily great, but if the loose tissue under the epicranial aponeurosis be affected, spread of the disease is certain to take place, and is limited only by the attachments of this membrane. The whole structure is liable to be raised off the surface of the skull by a collection of pus which, gravitating to dependent parts, is found more copiously collected over the eyebrows in front, just above the superior curved line of the occipital bone behind, and in the temporal regions at the sides. Owing to the large size

and short course of the veins connecting the intracranial venous sinuses with the veins of the scalp, septic sinus thrombosis is very liable to occur and lead to death.

Diagnosis presents few difficulties, but care should be taken not to mistake for cellulitis, and incise, the swellings on the dorsum of the hand which are the result of gout or gonorrhœa.

Treatment.—Absolute rest to the part is essential. The patient is put to bed, and if a limb is affected it should be raised on a pillow to aid the circulation. The pain should be relieved by hot boric-acid fomentations. Cold applications (evaporating lotions or ice-bags) should never be used; they may relieve pain, but they assist the activity of the organisms, and are thereby contraindicated. Early recourse should be had to incisions. Energetic treatment will cut short the disease where temporizing measures may be fraught with very serious consequences. It is not to be expected that pus will be evacuated by the incisions, of which several should be made in a direction parallel to subcutaneous nerves and veins. They should extend down to, but not through, the deep fascia—at least in cases where such treatment is adopted, as it should be, at an early stage. From the hard, brawny cut surface will exude serous fluid mixed with blood. Sloughs may be met with. In late cases the deep fascia must be cut through, and the infected spaces between the muscles opened up, drainage-tubes being left *in situ*. The free flow of serum and blood should not be stayed, but rather encouraged. Such exudation brings protective substances into contact with the infecting microbes. For this reason hot fomentations, frequently changed, or hot boric or iodine baths (tincture of iodine 1 dr. to water 1 pint) are useful in the after-treatment. Adopting another procedure, the wounds may be lightly filled with gauze kept soaked with a saturated solution of magnesium sulphate, which encourages exudation, inhibits clothing, and has an analgesic action; 5 per cent. sodium chloride solution may with advantage be used for saturating the gauze for the first three or four days—*Wright's salt treatment* (see WOUNDS, TREATMENT OF). A brisk purge is essential, and the patient should be induced to drink as much water or lemonade as possible. Stimulants may be necessary. Carrell's treatment (WOUNDS, TREATMENT OF), where it can be carried out, is excellent. Bier's passive congestion treatment

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I have not found to give the results claimed for it. It is, moreover, troublesome to carry out.

Vaccines should certainly be employed if possible, and an autogenous one is best, 5 million streptococci or 50 million staphylococci, or both, being given every three or four days. Very little is to be expected from the use of antistreptococcic serum. C. A. PANNETT.

CELLULITIS, ORBITAL (see ORBIT, AFFECTIONS OF).

CELLULITIS, PELVIC (see PELVIC CELLULITIS).

CEPHALALGIA (see HEADACHE).

CEPHALHÆMATOMA (see BIRTH INJURIES).

CEREBELLAR ABSCESS (see CEREBRAL ABSCESS).

CEREBELLAR ATAXY, HEREDITARY (see ATAXY, HEREDITARY CEREBELLAR).

CEREBELLAR TUMOUR (see CEREBRAL TUMOUR).

CEREBRAL ABSCESS.—Abscesses may be found in any part of the brain. They are due to the introduction of infective micro-organisms by a trauma that fractures or penetrates the skull; to the direct extension of infection from a neighbouring focus, or to its conveyance from a distant source by the blood or the lymph. The first group needs no further reference. Extension from an adjacent focus is unquestionably the usual cause in civil life. Bacteria may spread along the emissary vessels from a septic wound or erysipelas of the scalp, but chronic otitis media is much the most common source, and is probably responsible for more than half the cases. The ear disease may be recent, but it is more generally of long standing, or the abscess may follow an acute exacerbation, or the blocking up of pus by granulations. A cholesteatoma of the ear is occasionally the original lesion. When there is septic osteitis the disease may spread directly into the brain; this is liable to be the course when the thin bone of the roof of the tympanum is diseased, but as the dura mater forms a barrier to the further spread, an extradural abscess only may develop. Or the bacteria may extend along the penetrating vessels and lymph channels; the abscess is then often associated with thrombosis of the lateral sinus. Finally, pus from a suppurative labyrinthitis

may track along the internal auditory meatus. The type of the ear disease has an important influence on the site of the abscess; when the tympanum or the mastoid antrum is affected it generally lies in the temporal lobe, and in the cerebellum when the mastoid cells are involved.

Septic diseases of the nose and its accessory sinuses, and especially suppuration in the ethmoidal and frontal regions, is the next most frequent cause. The abscess then usually forms in the frontal or the anterior part of the temporal lobe. The majority of metastatic abscesses are secondary to purulent diseases of the lungs, particularly bronchiectasis, gangrene, and empyema. Occasionally the source is an ulcerative endocarditis or puerperal infection. These abscesses are often multiple.

Pathology.—There are two chief forms—circumscribed encysted collections of pus and diffuse purulent encephalitis. In the circumscribed form a definite wall develops from the inflammatory tissue, and eventually becomes so thick that it separates the abscess completely from the surrounding brain. In the diffuse type a central collection of pus is surrounded by an inflammatory zone, and this by an area of oedema. This type has an acute course, but those that become walled off are often chronic and may remain latent for years.

Symptomatology.—The **general symptoms** are those of increased intracranial pressure, i.e. headache, vomiting, a slowing of the pulse, and optic neuritis. The headache is generally very intense during the stage of development, and may correspond in position to the lesion; when this becomes stationary the pain is usually slight and intermittent. It is often referred to the back of the head, and may be associated with stiffness of the neck. Vomiting, which is associated with the severer bouts of headache, is of the ordinary cerebral type. Optic neuritis is less common than in cerebral tumour; there may be little more than hyperæmia and blurring of the discs with engorgement of the veins. A slow pulse is also inconstant; it may be rapid when there is much pyrexia. Stupor, delirium and coma appear as terminal symptoms. Convulsions are not very common.

The **local symptoms** depend on the destruction of cerebral tissue by the abscess, and vary with the site of the latter. Only the two most common types need be referred to here.

1. *Abscess of the temporo-sphenoidal lobe.*—This is a common complication of otitis media. It is generally situated in the inferior

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and posterior part of the lobe, which is one of the so-called "silent areas" of the brain, and definite localizing symptoms consequently appear only when other centres or fibres from them become involved. In the left hemisphere the speech centre may be affected so that signs of sensory aphasia develop. Inward extension or pressure that affects the internal capsule causes a partial hemiplegia or changes in the reflexes of the opposite side, but the most significant symptom is a slight weakness of the opposite side of the face and tongue, due to a compression of the pyramidal fibres from the lower part of the precentral gyrus as they arch over the temporal lobe to reach the internal capsule.

2. *Cerebellar abscess.*—Abscesses of otitic origin generally lie in the upper and anterior part of the cerebellum and produce symptoms similar to those of other unilateral lesions of this organ, i.e. ataxia, diminution of tone and paresis of the homolateral limbs, with an unsteady gait, a tendency to fall to the same side, and nystagmus on lateral fixation of the eyes which is coarser and slower on looking towards the affected side. It must be remembered that the symptoms of acute labyrinthitis may closely simulate those of cerebellar abscess, but though in it there may be tremor, there is no true ataxia, dysdiadochokinesis, or loss of power; nystagmus occurs apart from accurate fixation, and is more marked on lateral deviation towards the opposite side. Occasionally cerebellar abscesses produce no characteristic symptoms.

Diagnosis.—It is often difficult to be certain that there is an abscess, even with the development of intracranial symptoms, as these may be due to other conditions. *Meningitis* produces headache, vomiting, and even focal symptoms. Retraction of the head, stiffness of the neck, and Kernig's sign are not sufficiently distinguishing features, as they may occur with rise of intracranial pressure. The general state of the patient, the amount of fever, an increased pulse-rate, and particularly evidences of a diffuse lesion, as palsies of the cranial nerves, are more valuable indications. It is often necessary to do a lumbar puncture to be certain. *Serous meningitis* is frequently associated with septic processes in the neighbourhood of the brain, and especially with ear disease. All the signs of increase of intracranial pressure may be present, even optic neuritis, but focal symptoms are rare and the cerebro-spinal fluid remains clear. Examination of this often aids

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in the diagnosis, as when an abscess exists it frequently contains an increased number of cells, some of which are polymorphonuclear. There may be a leucocytosis in the blood also.

Treatment.—Cerebral abscess is a serious and frequently fatal condition. Operation and drainage as early as possible is the only justifiable procedure, but this should be preceded by exact diagnosis and accurate localization. The disastrous results of exposing and incising a healthy brain in the presence of a purulent infection must never be forgotten. The most satisfactory route is that by which the infection spread, if it is possible. The chief dangers of abscess are rupture into the lateral ventricle, a spreading septic encephalitis, and extension of the infection to the meninges, exciting a generalized meningitis. GORDON HOLMES.

CEREBRAL ANEURYSM (see CEREBRAL VASCULAR DISEASE).

CEREBRAL ARTERIO - SCLEROSIS.

Etiology.—Arterio-sclerosis of the brain is due to the causes of arterio-sclerosis in general, and yet sometimes the arteries of the brain are diseased with little or no evidence of an affection of the rest of the circulation. The condition is essentially one of old age. In the rare cases in which the cerebral vessels are affected in young people, hereditary syphilis, and even alcohol, have to be remembered as possible causes. Hereditary influences are also important.

Pathology.—No satisfactory explanation has been adduced why in certain cases the incidence of the disease should fall chiefly or even alone on the arteries of the brain. The primary changes in the vessels begin in the intima. The cells of this tunic, internal to the elastic lamina, proliferate and form nodular or diffuse thickenings. Secondary degenerative changes then occur, with fatty and calcareous metamorphosis, and the middle coat becomes involved. The openings of many of the smaller vessels into the main trunks are often occluded, and consequently minute thrombi occur. The brain tissue suffers from a lack of nutrition, and the disintegrative changes which accompany old age are hastened and accentuated.

Symptomatology.—Arterio-sclerosis may occasion the signs of cerebral hæmorrhage or thrombosis, as described in the article on CEREBRAL VASCULAR DISEASE, but apart from these gross manifestations there are important and often characteristic groups of symptoms

CEREBRAL DIPLEGIA

which indicate the presence of degenerative vascular disease in the brain. These are:

1. *Symptoms no doubt due to minute hæmorrhages or thrombosis.*—Such are sudden attacks of giddiness, vomiting, and momentary loss of consciousness, or slight signs of focal lesions, such as a very transitory aphasia, numbness and tingling in the hand or arm, or slight paresis of a limb.

2. *Epileptiform attacks, often partial and slight.*—These are sometimes followed by slight hemiplegia or monoplegia.

3. *Signs of mental decay,* and especially loss of memory for the events of more recent life, associated with lack of power of attention. The neurasthenia of old people may be a manifestation of vascular brain disease.

4. *The appearance is often suggestive.*—An immobile expression, which has been described as the inanimate replica of a normal person; a slowness in movement and a face pale in the morning, but red or bluish-red at night. A tendency to emotional attacks with meaningless laughter and weeping is not uncommon. In the daytime the patient is drowsy, often falling asleep, but is sleepless at night.

Headache is one of the most prominent symptoms; it is rarely severe. The patient generally refers it to the vertex or occipital region, and describes it as dull or throbbing, or occasionally as bursting pain. It is aggravated by exertion, worries, and emotions.

Diagnosis.—The age, the symptoms of the disease, and perhaps evidence of vascular disease elsewhere, give the diagnosis. There is no optic neuritis, and usually no definite paralysis. A blow on the head in such persons may complicate the diagnosis. The common mistake made is in not recognizing the serious nature of comparatively slight symptoms, which are often considered as a mere "neurasthenia."

Prognosis and treatment.—The prognosis is bad, and but little can be done to prevent the onward march of the disease.

W. B. WARRINGTON.

CEREBRAL COMPRESSION (*see* HEAD INJURIES).

CEREBRAL CONCUSSION (*see* HEAD INJURIES).

CEREBRAL DIPLEGIA.—This condition may be due to many different pathological lesions, and includes cases with widely varying symptoms, but all are characterized by signs

of bilateral cerebral lesions which arise in intra-uterine life, during birth, or in early infancy.

Etiology.—Many of the cases that date from prenatal life are due to cerebral aplasia or non-development of the brain; this form is frequently familial, and may be associated with premature birth. Others appear to be infectious in origin, or the result of a syphilitic meningo-encephalitis. A second group is due to mechanical injuries at birth, which have caused meningeal and cerebral hæmorrhages, or even extensive damage to the brain. Over-riding of the parietal bones may tear the cortical veins as these enter the longitudinal sinus and thus produce superficial hæmorrhages which compress and interfere with the growth of the cortex. Asphyxia neonatorum may be responsible for some of these cases. In a third group, which has frequently an hereditary or a familial incidence, degenerative changes, probably due to developmental defects in the tissues, set in after birth. Other postnatal cases are the result of encephalitic lesions occurring during the course of one of the specific fevers.

Pathology.—The morbid anatomy necessarily varies with the cause. The most common pathological change is a diffuse sclerosis of the cerebral hemispheres. The convolutions may be small and irregularly arranged (*microgyria*). In other cases there is a defective development of a part of the brain which is generally symmetrical, or there may be porencephalic cavities which communicate with the ventricles in one or both hemispheres. The meninges may be thickened or contain cysts. Histological examination usually reveals small, poorly developed and irregularly arranged nerve-cells in the cerebral cortex, with an excess of neuroglia and defective development of the pyramidal tracts.

Symptomatology.—The commonest clinical variety is that known as *Little's disease*. This presents extreme rigidity of the lower limbs, associated with a varying degree of weakness. The face and upper limbs may escape, or at least are less severely involved than the legs. These symptoms may be noticed very soon after birth, but in some cases attention is drawn to the condition by the fact that the child begins to walk late and then is clumsy. The legs are usually extended and strongly adducted and rotated inwards, with the knees in apposition and the ankles and feet crossed. On attempts to walk this tendency is increased and produces the familiar "scissors gait." Walking without support may not be possible.

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Commonly the trunk and head are bent forwards, and the patient shuffles along as though about to fall forward on his face. Contracture of the calf muscles prevent the heels from being put to the ground, and gives a "digitigrade" character to the gait. Eventually, contractures tend to develop and to fix the vicious attitudes described. There is rarely much loss of power in the arms, though they may be rigid, thus producing a clumsiness of fine movements. The knee- and ankle-jerks are increased, clonus can be elicited, and the plantar response is of the extensor type.

In one group, involuntary movements are the predominant symptom; these may take the form of athetosis, or choreiform jerking, or movements with features of both these types. In the athetosis group the arms are chiefly affected, and there may be no rigidity, weakness, or alteration of the reflexes, such as are found in Little's disease.

Speech is usually impaired; there may be dysarthria of the type seen in pseudo-bulbar paralysis, or the patients are merely slow in learning to talk, and acquire only a limited vocabulary. All grades of *mental defect*, from the slightest to complete idiocy, occur, but many children show a marked aptitude in certain respects, as retentive memories or extraordinary powers of calculation. A large proportion of the patients suffer with *epilepsy*; the fits are usually generalized, but occasionally are unilateral or of the Jacksonian type. The seizures as a rule begin in early childhood, and in many cases cease or become less frequent about puberty. Often they persist and lead to an epileptic dementia. *Blindness*, due to optic atrophy or to choroidal defect, may be present, and squint and nystagmus are common. Certain *deformities*, such as talipes, scoliosis, and contractures of the limbs are frequently seen in longstanding cases.

Prognosis.—This is bad as far as improvement is concerned. Some cases become steadily worse, while others remain stationary throughout life. Many of the patients die in childhood from intercurrent disease, while of those who survive longer some become confirmed epileptics or idiots.

Treatment.—The cases of Little's disease with good voluntary power and no mental defect offer the best prospects of amelioration. For the others little can be done. Massage, active exercises, reinforced but not replaced by tendon stretching and judicious tenotomy, will often effect considerable improvement in

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the patient's comfort and mobility. In favourable cases Förster's operation of posterior-root section may free the patient from the hampering effects of rigidity. It is essential that for this operation cases with good voluntary power should be chosen, but since it is one of some difficulty and danger, its application is obviously limited. Cases due to delayed or arrested development of the brain occasionally improve after the administration of thyroid and other organic extracts.

F. M. R. WALSHE.

CEREBRAL EMBOLISM (*see CEREBRAL VASCULAR DISEASE*).

CEREBRAL HÆMORRHAGE (*see CEREBRAL VASCULAR DISEASE*).

CEREBRAL MENINGITIS (*see MENINGITIS*).

CEREBRAL SCLEROSIS.—This is a pathological rather than a clinical entity, and, even so considered, includes several conditions differing widely etiologically.

A *diffuse* cerebral sclerosis is the common lesion of cerebral diplegia. In these circumstances it may be either bilateral or confined (in infantile hemiplegia) to a single hemisphere. The convolutions are shrunken, the sulci wide and gaping. There is marked deficiency in the number of fibres present, and the basal ganglia may be shrunken also. The pyramidal tracts show defective myelination or development.

The name is applied also to a progressive disease found usually in children, but also in adults, and characterized clinically by progressive general paralysis and dementia, passing into stupor. Post mortem, the white substance of the hemispheres is tough and leathery in consistence, the pons and medulla are shrunken. The microscopical appearances are not characteristic and are minimal. Clinically, such cases are difficult of diagnosis, and have to be differentiated from the commoner disseminated sclerosis and dementia paralytica, or from the rare pseudo-sclerosis. Congenital syphilis and head injuries have been thought to underlie this condition.

Localized cerebral sclerosis may follow embolism or thrombosis, and is then limited to the distribution of the cerebral artery affected.

A well-defined but obscure condition is *tuberculous sclerosis*, in which nodules of glial cell tissue are found in the substance and on the surface of the hemispheres. Accompanying there are nodular tumours in the viscera varying in

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structure according to the organ in which they occur. Clinically these cases are characterized by idiocy and epilepsy. The condition is thought to be developmental and to date from intra-uterine life.

F. M. R. WALSHE.

CEREBRAL SINUSES, THROMBOSIS OF.—Thrombosis of the cerebral sinuses other than the lateral sinus (*see* LATERAL-SINUS THROMBOSIS) is not very common. The other sinuses most frequently involved, at least as far as is recognizable from symptoms, are the superior longitudinal and the cavernous.

Thrombosis may occur under two conditions. In the first group of cases there is some definite local starting-point, as the pressure of a tumour or a depressed fracture, or it is secondary to inflammation in some contiguous spot. In the other group there is no discoverable local cause, but the patients are usually very young or very old persons in whom the circulation is feeble, or who are the subjects of wasting diseases, as phthisis, chronic diarrhoea or carcinoma. Thrombosis occurring in these circumstances is spoken of as marantic.

Symptomatology.—The symptoms are usually vague and are often masked by those of an accompanying meningitis, especially when thrombosis is secondary to a local infection. General symptoms are headache, restlessness, delirium, convulsions, and coma.

The superior longitudinal sinus is most frequently affected by marantic thrombosis, but it may become thrombosed secondarily to inflammation in the nasal passages. In addition to general symptoms this gives rise to epistaxis and to venous congestion and oedema at the top of the head. Both are due to the obstruction of extracranial veins which, passing through the skull, drain into that sinus. In addition, there may be some paresis in the limbs owing to blockage of the venous return from the cortex cerebri.

Thrombosis of the cavernous sinus may follow septic inflammation in the orbit or in the region of the nose, e.g. erysipelas. The chief symptoms are oedema of the eyelids and upper part of the cheek and nose, and conjunctival ecchymoses; there may be also exophthalmos and paralysis of various ocular muscles due to involvement of the ocular nerves as they pass through the wall of the sinus.

Diagnosis.—Congestion of extracranial veins is the most trustworthy guide in diagnosis, as obviously many intracranial conditions

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other than thrombosis may cause all the cerebral symptoms.

Treatment.—In cases of secondary thrombosis the original septic focus should be removed or drained, and an attempt should be made to deal directly with the thrombus in the sinus by surgical means. Of marantic thrombosis the treatment is symptomatic. The prognosis is very unfavourable but not hopeless.

F. C. PURSER.

CEREBRAL SYPHILIS (*see* CEREBRO-SPINAL SYPHILIS).

CEREBRAL THROMBOSIS (*see* CEREBRAL VASCULAR DISEASE).

CEREBRAL TUMOUR.—There is probably no organ in the body which, in relation to its size, is so frequently invaded by tumours as the brain, and there can be no doubt that in a large proportion of the cases the condition is not recognized during life.

Cerebral tumour may occur at any age from early infancy to late life, but is most frequent in the adult years. Many different forms occur; the relative frequency of each varies with age and the region of the brain involved. The most common is a *glioma*, which is a sarcomatous tumour that takes origin from the neuroglia and infiltrates the surrounding portions of the brain. Gliomata are generally very vascular, and as their vessels have thin walls, hæmorrhage frequently takes place into them; or if the blood supply fails they may undergo central necrosis, the necrotic material being gradually absorbed and replaced by a serous or gelatinous fluid so that a cyst develops; alterations of pressure within this may lead to sudden exacerbations or changes in the symptoms. *Sarcomata* which grow from the mesoblastic tissue of the vessels or meninges are also infiltrating tumours, but are less common. *Tuberculous tumours* are particularly frequent in children, but occur in later life too. They are generally attached to the meninges, and lie superficial in the forebrain, but are found also in the substance of the pons, medulla, and cerebellum. *Gummata* too are nearly always superficial, as they usually arise from the meninges. One important and not rare tumour is the *endothelioma* or *psammoma*, which develops from the endothelium that lines the under surface of the dura mater. As it merely compresses the brain, and is separated from it by the soft meninges, it can be removed with complete success. *Fibromata* grow

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chiefly from the cranial nerves, especially from the auditory. They often undergo myxomatous change. *Carcinomata* are the most common of the metastatic growths. The brain may also be compressed or invaded by tumours originating in the skull.

Etiology.—We know as little of the cause of tumour growth in the brain as in any other organ. A certain proportion of the gliomata and other tumours originating in the nervous tissue are probably due to developmental anomalies. In a considerable number of cases the symptoms set in after an injury to the head; it is possible that the trauma has some influence on their genesis, but on the other hand it may merely serve to draw attention to symptoms hitherto so slight as to pass unnoticed.

Symptomatology.—The course varies enormously. Some tumours progress to a fatal termination within a few months, in others the symptoms may last for years. This variability depends largely on the nature of the growth. Gliomata often grow rapidly while endotheliomata increase very slowly. Gummata and tuberculous tumours may become stationary.

The symptoms of intracranial growths are general and local. The **general symptoms**, which are independent of the nature and position of the tumour, are due to an increase of intracranial pressure. They are obviously not pathognomonic of tumour, as they may result from abscess, hydrocephalus, or any other cause of increased pressure. *Headache* is the most common and constant. Its character is usually that of a deep boring, expanding or throbbing pain, which is more or less continuous, but is increased by exertion or anything that raises the blood-pressure or tends to congest the brain. It is often most severe in the early morning, or it may increase during the day and subside when night brings rest and quiet. Its position varies; when the tumour is superficial it may be most intense over it and associated with tenderness of the skull in this region; in other cases it is general, or of variable intensity in different parts of the head. Persistent headache above and behind the eyes is very characteristic in forebrain tumours, while those that occupy the posterior fossa tend to produce occipital or suboccipital pain, often with stiffness of the neck.

Vomiting is an important symptom, though not so common as is generally assumed. It is of the cerebral type—the contents of the

stomach are suddenly projected through the mouth without, as a rule, any nausea or pain. It is usually associated with intenser bouts of headache, and is often only a late symptom. Early morning vomiting as the patient rises from bed should, in the absence of pregnancy, excite the suspicion of intracranial disease. It is most common with tumours of the posterior fossa.

Optic neuritis, a condition of cedema and congestion of the optic discs and hence better called *papilloedema*, is a still more valuable sign, as with rare exceptions its typical form always denotes a pathological rise of intracranial pressure. It must be emphasized that at first it produces no deterioration or subjective disturbance of vision, and that sight fails only when it subsides into secondary atrophy. Its earliest appearance is merely a hyperæmia of the discs with congestion of the retinal veins; later, swelling of the nerve-head, hæmorrhages and retinal changes develop. But papilloedema is by no means constant. It may appear only late in the history of a case, or never develop. This depends on the nature, site, and rapidity of the growth. It is common in gliomata, carcinomata, and rapidly growing tumours, rarer in tuberculous and syphilitic lesions. Its absence is the rule with tumours of the pons, while it develops early and is almost invariable with neoplasms of the cerebellum and midbrain.

A *slow pulse-rate* is a sign that has received an exaggerated importance. It is frequently never worthy of note, and is usually pronounced only in the later stages, or when an associated hydrocephalus increases the intracranial pressure rapidly. A *high blood-pressure* for which no other cause can be discovered is often seen; it is due to the effort of the cardio-vascular system to give a sufficient supply of blood to the brain despite the resistance which the flow encounters from the rise of intracranial pressure.

Mental changes, as dullness, drowsiness and apathy, are not uncommon, quite apart from the local effects of the lesion. In pronounced degree they are usually late symptoms, and more or less parallel to and dependent on the rise of pressure. Drowsiness often passes into stupor, and this into a terminal coma.

Local symptoms.—These, which are due to destruction or compression of the part of the brain that is involved, naturally vary with the site of the tumour. When the growth invades one of the so-called "silent areas" there may

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be no local symptoms, or signs can be detected only by careful and expert examination. The local signs of tumour present, as a rule, features that are often indicative, or at least suggestive of their origin. The first is the slow and gradual onset of the symptoms. While vascular lesions and inflammatory foci affecting the motor centres or fibres produce a rapidly developing weakness which tends to recover, a tumour in the same site causes a more gradually progressive disturbance of function; the weakness increases *pari passu* with its growth, and this is usually slow. There are, however, exceptions to this rule; in old arterio-sclerotic patients a progressive thrombosis may be responsible for a gradually increasing hemiparesis or other symptom, while the loss of function due to tumour may be more or less sudden—when, for instance, a hæmorrhage occurs into a glioma or the tension within a cyst suddenly rises. A sudden hemiplegia may also follow convulsive seizures due to the irritative effects of a tumour.

In the second place, while vascular and other lesions generally produce a definitive state, the symptoms of a neoplasm which are at first usually local become gradually more widespread. Thus a tumour near the lower end of the fissure of Rolando may cause at first only weakness of the face, but as it grows the arm, and later the leg, may become weak too. In a patient under my care the first symptom was a palsy of the left sixth nerve, then weakness of the conjugate movements of the eyes to the left appeared, next a left facial palsy, later motor and sensory trigeminal symptoms, and after four months sensory disturbances on the right side of the body. This slow progress, obviously determined by the anatomical arrangement of the different structures, can be due only to a lesion that increases slowly in size; one of vascular origin or an acute infection could not produce it.

Finally, tumours are very liable to cause irritative phenomena, as motor convulsions, transient sensory or visual disturbances, or generalized epileptic fits which generally commence with a local aura.

It is beyond the scope of this work to deal with the exact localization of cerebral tumours, but the usual features of growths in some of the more important and common regions may be described shortly.

Tumours of the Rolandic region.—The local symptoms are due to an interference with

motor centres which produces weakness of the opposite side of the body; as a rule this begins locally, for instance, in the face, hand, or foot, and spreads gradually. The paresis is similar to that of hemiplegia, and is associated with the same changes in the reflexes, but spasticity develops slowly or not at all. When the lesion extends behind the central fissure there are disturbances of sensation too, chiefly of the discriminative forms, as loss of the sense of position, astereognosis and defective localization. If the growth lies in or near the cortex, localized or Jacksonian convulsions often affect the opposite limbs; these may remain local, limited to the hand or fingers for instance, or, following a definite march, involve all the muscles of the opposite side; or they may lead to generalized convulsions in which the contralateral limbs are always most affected. The place of onset of these spasms is a valuable guide to the actual site of the tumour; they begin, as a rule, in the face when it is in the lower part of the motor region, and in the foot or leg when it is situated near the apex of the hemisphere. Its position in relation to the central fissure is also indicated by the first symptom of an attack; clonic movements occur first when it is in the frontal lobe, while sensory phenomena, as numbness or tingling, are the initial symptom when the growth is behind the fissure of Rolando or in the parietal lobe.

Tumours of the frontal lobe.—Lesions here may produce no distinctive features, and it is consequently difficult to localize tumours of this region with certainty. Backward pressure is, however, liable to affect the motor centres and cause a contralateral hemiparesis or weakness of one limb, with changes in the reflexes, but without sensory disturbances. Absence or diminution of the opposite abdominal reflex is often the earliest local sign. Involvement of Broca's area affects motor speech, and as the frontal lobes also contain centres for the conjugate movements of the eyes, deviation to the opposite side may be incomplete or require more effort. Two types of convulsions occur—Jacksonian fits, which almost always begin with rotation of the head and eyes towards the opposite side, and generalized epileptic seizures without local auras. Tumours in the basal part of this lobe may compress the olfactory tract and cause loss of smell in the homolateral nostril, or the optic nerve and reduce the visual acuity of this eye. Then papilloedema does not develop, or is less than on the opposite side.

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The mental symptoms are frequently suggestive, if not characteristic. Some patients become dull and listless, and pass into a childish state suggestive of early dementia; in others the chief feature is a lack of inhibition and reserve, shown in impulsive acts, unreliable temper, or facetious remarks which are irrelevant or out of place.

Tumours of the parietal lobe.—Sensory disturbances, especially astereognosis and loss of the sense of position, combined with paresis of the opposite limbs, are the most constant symptoms when the growth lies in the anterior part of the lobe. When in its inferior part and on the left side various forms of sensory aphasia occur. Tumours in the region of the angular gyrus are liable to produce hemianopia by pressure on the optic radiations; if they occur in the neighbourhood of the supramarginal gyrus apraxia may result, especially when the growth extends deeply into the lobe.

Tumours of the occipital lobe.—Here the characteristic symptom is a disturbance of vision in the form of a hemianopia, hemiamblyopia, or scotoma due to involvement of the optic radiations or visual cortex in the calcarine region. Subjective visual sensations, as flashes of light or the appearance of simple figures, which may be either coloured or uncoloured, in the opposite hemiopic fields, may be due to irritative lesions near the calcarine cortex. They are very important signs. No motor or sensory disturbances occur.

Tumours of the temporo-sphenoidal lobe.—Lesions in the right temporal lobe do not produce recognized localizing symptoms, but in the left lobe disturbances of sensory speech, which may be only a slight difficulty in recalling names, are common when the lesion lies in the upper part of the lobe. Sensory symptoms are rare unless the growth extends deeply towards the internal capsule or optic thalamus. In many cases there is no motor weakness or alteration in the reflexes, but as the corticobulbar fibres from the lower end of the precentral gyrus pass almost horizontally inwards towards the internal capsule they are often compressed by tumours in the dorsal part of the lobe, so that some weakness of the opposite side of the face and tongue results. The most characteristic symptom, however, is the occurrence of "uncinate fits;" these commence with gustatory or olfactory auræ, mostly an unpleasant smell or taste, and are sometimes succeeded by the "dreamy state" described by Hughlings Jackson, in which the patient

remains conscious but feels himself in other surroundings. Irregular clonic spasms or unilateral convulsions of the opposite limbs may develop. These uncinate fits are, however, associated only with lesions of the inner part of the lobe.

Tumours in the depth of the forebrain.—It must be admitted that we are often unable to determine by physical signs whether a tumour is situated superficially or deeply in the brain. Some of the points on which a diagnosis can be based have been already considered. Other indications of a deeply situated growth are a gradually developing hemiplegia that from the onset affects leg, arm, and face more or less equally, owing to compression or invasion of the internal capsule. It is usually combined with sensory disturbances much grosser than those due to cortical lesions, and occasionally with hemianopia. Local or Jacksonian spasms are rare, but general seizures may occur. Other important localizing symptoms are due to pressure on the cranial nerves at the base of the brain; there may be hemianopia with Wernicke's hemiopic pupillary reaction when the optic tract suffers; loss of taste when the olfactory tract is compressed; pain in the same side of the face, with anæsthesia and perhaps neuropathic keratitis and weakness of the masticatory muscles, if the trigeminal nerve is involved; or diplopia. Symptoms of pressure on the midbrain occur frequently; these are usually inequality and reflex inactivity of the pupils with weakness of certain ocular muscles, especially of those concerned in upward rotation of the eyes. The general symptoms, too, are suggestive; the headache is more diffuse, the pulse is as a rule slower, and as a rule optic neuritis does not develop so early as when the tumour is superficial.

Tumours of the midbrain.—These are characterized by diplopia and weakness of the homolateral ocular muscles, associated with contralateral hemiparesis. A mesial tumour in the anterior part of the midbrain is liable to produce pupillary anomalies with loss of the vertical movements of the eyes. There may be ataxia of the limbs of one side, or a tremor resembling that of paralysis agitans. Bilateral bulbar symptoms, as dysarthria or dysphagia, often appear. The headache is generally very severe and papilloedema early and intense, and the symptoms of increased intracranial pressure are marked, internal hydrocephalus being common.

Tumours of the pons and medulla oblongata.

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—Lesions in this region produce alternate hemiplegias, the cranial nerves of the same side and the limbs of the other side being affected. In the upper part of the pons the trigeminal suffers, so that there are pain, anæsthesia, palsy of the masticatory muscles and often keratitis; in its lower portion it is chiefly the facial and abducens nerves that are paralysed, or when the nucleus of the sixth nerve is involved there is loss of conjugate movement of the eyes to the same side. In the medulla the nerves that innervate the palate, pharynx, and tongue chiefly suffer. Pontine and medullary tumours frequently produce homolateral ataxia and other symptoms of cerebellar disease, as the afferent or efferent connections of the cerebellum may be disturbed. Optic neuritis is late or may never develop, especially with pontine growths.

Tumours of the cerebellum.—Tumours of one lateral lobe produce disturbances of cerebellar function on the same side of the body—that is, nystagmus which is slower and coarser on deviation of the eyes to that side, loss of tone, inco-ordination or tremor with feebleness of the homolateral limbs, slowness and clumsiness in performing rapid alternate movements, and deviation of the arm outwards in Bárány's pointing test. The disturbance of gait is often the most pronounced symptom; the rule is that the patient inclines to the affected side and is in danger of falling to it when standing; in walking he staggers, reels, and deviates to this side, and it can be often seen that he places the homolateral foot irregularly. Central or bilateral tumours cause bilateral symptoms and a drunken staggering gait, often with a tendency to fall backwards or forwards. Vertigo is a common symptom, but as it may be due to tumours in any region of the brain, too much weight must not be given to it as a localizing sign. Papilloedema almost invariably occurs early, and the headache, which is usually intense, is situated at the back of the head and neck.

Extracerebellar and auditory-nerve tumours.

—The cerebellum may also be compressed by tumours growing from other structures in the posterior fossa. The most common of these are tumours of the auditory nerve. They are generally fibromata. Tinnitus and progressive deafness of one ear are the first symptoms; later, weakness of the same side of the face appears, often associated with pain due to compression of the trigeminal nerve. Absence of the corneal reflex is usually the earliest

indication of this. The external rectus is frequently paralysed too, and there may be a relative weakness of one side of the palate and loss of the palatal reflex. The symptoms of cerebellar compression are similar to those due to cerebellar tumours. Occasionally tumours grow simultaneously on both auditory nerves.

Differential diagnosis.—It is not always easy to be certain of the existence of a cerebral tumour, even in the presence of the three cardinal signs—headache, vomiting, and papilloedema. Of these, papilloedema is the most important, but it is essential to make sure that it is the true papilloedema that results from intracranial pressure, and not renal or arterio-sclerotic neuro-retinitis, or the swelling of the disc that is sometimes associated with retrobulbar disease. In these conditions sight, and especially central vision, is affected early.

The distinction from *cerebral abscess* may be impossible, as both conditions produce the same symptoms. The presence of a possible focus of infection and a relatively rapid progress are suggestive of abscess. Examination of the cerebro-spinal fluid may help, as when an abscess is present it frequently contains an excess of cells, and particularly polymorphonuclear leucocytes. In *syphilitic meningo-encephalitis* papilloedema is rarer, the headache is frequently local and superficial, scattered cranial-nerve palsies and pupillary anomalies are common, and examination of the blood and cerebro-spinal fluid makes the diagnosis certain.

In *cerebral meningitis* the cranial nerves also suffer, the course is more rapid, and there are generally fever and such symptoms of meningitis as pain and stiffness of the neck and Kernig's sign. *Serous meningitis* which produces symptoms of intracranial pressure with or without localizing signs is more difficult and often impossible to exclude. It occurs generally in the course of an infection or of a local collection of pus; its progress is more rapid and intermittent, and it is frequently relieved by lumbar puncture.

All the symptoms of an intracranial neoplasm occasionally subside and never recur. This condition has been called *pseudo-tumour*. The nature of the lesion is doubtful; in some cases it may be a serous meningitis or a local swelling of the brain around an infective or irritant focus. Recovery has been attributed to large doses of potassium iodide. But the most common source of confusion is *renal disease*, especially when associated with cerebral arterio-

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sclerosis. The headache, which is more or less constant, is, like that of cerebral tumour, increased by exercise and movement; vomiting or retching occasionally occurs, and the albuminuric or arterial changes in the fundus may be mistaken for papilloedema. The presence of albumin in the urine and a high blood-pressure should excite suspicion, and the ophthalmoscopic appearances can be distinguished by careful examination. Further, when local signs occur they appear suddenly and tend to improve. The persistent headaches of *neurasthenia* may occasionally suggest a cerebral tumour, but the character of the pain is quite different; it is usually a feeling of weight or constriction of the head, is superficial and often associated with tenderness of the scalp, is increased by emotion and worry, and disappears when attention is diverted from it. If vomiting occurs it has not the character of that due to cerebral disease, and there is never any ophthalmoscopic abnormality.

Treatment.—Tuberculous tumours occasionally become latent and produce no further symptoms, gummata as a rule respond to energetic antisyphilitic treatment, but all other tumours are uninfluenced by medical remedies and have a progressive course to a fatal termination if unrelieved. Surgical removal is the only reasonable hope, but success can be expected only when accurate localization is possible. The onus of failure and the fame of success belong to the physician who directs the surgeon.

But the proportion of cerebral tumours that can be removed is relatively small, as many produce no signs by which they can be localized, some are inaccessible, and others infiltrate the brain so widely that it is impossible to extirpate them. The treatment of these inoperable tumours is, however, important. By making a large opening in the skull and decompressing the brain, we can at least relieve the headache and check the distressing vomiting. Further, decompression arrests the optic neuritis, if resorted to before secondary atrophy supervenes, and preserves sight; this is important, for if the tumour is slow-growing, or if it undergoes retrogressive changes, the patient may live for years. Early operation to save sight, regardless of the localization and possibility of removing the tumour, has become dangerously popular; it must be remembered that even intense papilloedema may persist for long periods without danger to vision, and that a decompressive operation may cloak the

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localizing signs that develop later and consequently make a radical operation impossible.

In the absence of localizing symptoms, or when the tumour is inaccessible, a right subtemporal decompression is the most advisable procedure, but if the tumour can be localized the trephine opening should be made above it, as we are always uncertain of its nature till it is exposed, and even partial removal of an infiltrating growth is often surprisingly successful.

The danger of lumbar puncture must also be emphasized; sudden death may occur after the removal of any large amount of cerebrospinal fluid; if some is required for diagnostic purposes, only a few drops should be taken.

GORDON HOLMES.

CEREBRAL VASCULAR DISEASE.—

Under this head are considered Cerebral Hæmorrhage, Cerebral Embolism, and Cerebral Thrombosis. Cerebral Arterio-sclerosis is considered under that title.

CEREBRAL HÆMORRHAGE.—Statistics show that though cerebral hæmorrhage may occur at any age, it is relatively more common in the fifth, sixth, and seventh decades. It rarely occurs apart from pre-existing disease, but every now and then fatal cases appear in which post-mortem examination fails to reveal any associated lesions.

Etiology.—The predisposing causes are high blood-pressure and disease of the cerebral arteries, and as these two conditions are associated with chronic renal disease it is not surprising that this is found in about one-third of all the cases. Syphilis and infective endocarditis are other important causes of cerebral vascular degeneration. Cerebral hæmorrhage may also occur in blood diseases, as in leukæmia, or into a tumour of the brain. Certain families show an hereditary tendency to cerebral hæmorrhage, generally due to hereditary arterial disease.

Pathology.—One of the commonest causes of hæmorrhage is rupture of a *miliary aneurysm*. These vary in size from a pin's head to a pea, and are most frequently found on the minute twigs that penetrate the basal ganglia. They are due to the high blood-pressure of renal disease, or to weakening of the vessel-walls by syphilitic or infective lesions. Hæmorrhage may, however, occur directly from diseased arteries; this is frequently due to atrophy of the brain around

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arterio-sclerotic vessels, and as these then lose the support that the brain tissue naturally gives them, they are liable to rupture on any sudden increase of the blood-pressure.

The most common site of hæmorrhage in the brain is in the neighbourhood of the internal capsule; here it is one of the lenticulo-striate branches of the middle cerebral artery that usually ruptures, but hæmorrhage may occur in any region of the brain. The extravasated blood destroys the nervous tissues in the neighbourhood of the rupture and produces œdema, circulatory disturbances, and an inflammatory reaction in a wider zone around it. Neurological and fibrous scar tissue that is later laid down in this zone eventually walls off the damaged portion. Occasionally the blood bursts into one lateral ventricle; a primary ventricular hæmorrhage from the choroid plexus is rare. Meningeal hæmorrhage is usually of traumatic origin (*see HEAD INJURIES*), but may be due to rupture of an aneurysm on a vessel at the base of the brain.

The post-mortem appearances vary with the age of the hæmorrhage; at first the blood is only partly coagulated, but within a few days the clot becomes firmer and assumes a chocolate colour. Reactionary changes commence around this apoplectic area, and a fairly well defined wall is thus formed, which contains a dark-coloured fluid, or if this is absorbed nothing but some scar tissue may remain.

Symptomatology.—The classical symptom of a cerebral hæmorrhage is an apoplexy—a state of profound unconsciousness of more or less sudden onset. The onset of the apoplexy varies considerably; it may be absolutely sudden and without warning, but the patient occasionally calls out with a pain in his head before he sinks into unconsciousness. Sometimes more definite prodromata, as giddiness, vomiting, numbness, transitory difficulty in speech, or a disturbance in vision, precede the attack. Or a definite paralysis may be the first symptom. Unconsciousness occasionally develops slowly and passes gradually into coma—*in-gravescent* apoplexy. Convulsions are not common, but a unilateral rigidity or flaccidity of the limbs, indicating the onset of a hemiplegia, can often be observed. If hæmorrhage into the ventricles or pons occurs there may be a universal rigidity; or if at the base of the brain, tetanus-like seizures with opisthotonos. A conjugate deviation of the head and eyes is common; it is towards the side of the lesion when this is destructive, and is then due

to the unbalanced action of the oculo-motor centres in the intact hemisphere; but when convulsions occur the eyes turn away from the side of the lesion.

The state of the reflexes varies; the knee-jerk of the affected side is often lost at first but is later increased; ankle-clonus can then be obtained, and stimulation of the sole evokes an extensor response. The abdominal reflexes of this side are diminished or disappear. The pupils are generally of medium size, but with pontine hæmorrhage they become small. The temperature is usually subnormal at the commencement of the attack, but rises to the normal or above it within 24–48 hours.

The symptoms of apoplexy are due to compression of the arteries of the medulla by the increase of intracranial pressure. It may be divided into four stages. In the first there is perfect circulatory compensation; the pulse and respiration are little disturbed, and the patient recovers quickly from coma. In the second, signs of compression begin to appear; the blood-pressure rises, the pulse becomes stronger, and respiration slower and deeper. In the third stage these symptoms reach their height, and the full picture of apoplexy develops. In the fourth the bulbar centres can no longer struggle against the increasing cerebral anæmia; the blood-pressure falls, the pulse becomes rapid and feeble, and the respirations shallow. If consciousness returns, disturbances in the functions of the central nervous system usually become apparent. Hemiplegia and the other symptoms that ensue are described elsewhere (*see HEMIPLEGIA*).

CEREBRAL EMBOLISM.—Emboli are most commonly derived from the valves of the heart. They are more frequent in the ulcerative than in the simple form of endocarditis, and are then generally derived from the aortic valve. Of simple valvular lesions, mitral stenosis is the commonest cause of embolism, the embolus being detached from a thrombus in the left auricle. Other sources are clots in the left auricle, or aneurysms or atheromatous patches in the aorta. Emboli may also come from pulmonary clots, and as the foramen ovale occasionally remains patent, those detached from the peripheral veins may pass from the right to the left ventricle and thence to the brain. Rarer forms are fat emboli from bones, masses of tumour cells, bubbles of gas as in caisson disease, and of air introduced into the veins.

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Embolism and thrombosis of cerebral vessels lead to an anæmia of the nervous tissue within the territory of their supply, and as the branches of most of the cerebral arteries are terminal and do not anastomose, an infarction follows. The colour of the infarct depends on the amount of blood within it; it may be red or hæmorrhagic, or pale yellow or anæmic.

The **symptoms** of cerebral embolism come on abruptly and usually without warning. Consciousness is rarely lost when the emboli are small; but when they are larger, and especially when they come from an infective endocarditis, convulsions and a rapidly deepening coma may ensue, and the condition becomes that of an apoplexy.

CEREBRAL THROMBOSIS.—The factors determining thrombosis are disease of the blood-vessels, feeble action of the heart, and states of the blood that predispose to coagulation.

Syphilitic endarteritis and arterio-sclerosis are the most important causes. Syphilis produces a thickening of the walls of the vessels, and particularly of their inner coats, that may even lead to their complete occlusion. It is most frequent in young adult males, and manifests itself generally within a few months to a year of the primary infection. The roughness and irregular calibre of the vessels in arterio-sclerosis also predispose to thrombosis, and particularly when the openings of the smaller vessels are involved. A feeble heart with low blood-pressure, such as is due to disease of the myocardium, may contribute to arterial clotting, especially if there is also some arterio-sclerosis. In some cases, however, no definite cause can be discovered; and it is probable that the thrombosis is a result of an altered blood state. These cases are most common in women in poor general health.

Symptomatology.—In syphilitic cases not seldom there are warnings that the cerebral vessels are diseased. Headaches and such signs of slight brain lesions as transitory weakness of an arm or leg, slight difficulty in speech, or verbal amnesia, are important indications. The onset of the paralysis may not be quite sudden, and there is rarely profound coma. Thrombosis of the basilar artery, which is rare and generally due to syphilitic endarteritis, produces paralysis of all four limbs and early loss of consciousness. The prognosis is very grave.

Thrombosis due to arterio-sclerosis is often preceded by similar prodromal signs. In slight

cases consciousness may not be lost, but in extensive lesions there is generally deep coma and a condition resembling the apoplexy of cerebral hæmorrhage. Recurrences are frequent, occasionally taking place at short intervals of a few days. A chronic progressive softening of the brain, due to slowly extending multiple thrombosis, may produce a gradually increasing paralysis, often associated with signs of irritation, and may resemble a cerebral tumour.

Diagnosis of cerebral vascular disease.

—When confronted with an unconscious patient the question of cerebral hæmorrhage has usually to be considered. The apparent age of the person at once introduces a bias, to which, however, undue weight must not be given. The possibility of epilepsy and of poisoning by narcotic drugs, as opium, alcohol, chloral, chloroform, and carbolic acid must be remembered. Apoplectiform attacks also occur in the course of general paralysis. Signs of traumatism are often open to ambiguous interpretation, as the injury may be either the cause or a result of the fall. An interval of recovery of consciousness before coma sets in is characteristic of hæmorrhage from rupture of the middle meningeal artery, but such temporary recovery occasionally occurs in spontaneous intracerebral hæmorrhage.

In the examination of the patient attention should be given especially to the following points:—

1. Signs of injury to the skull, escape of blood and fluid from the nose or ears, or the presence of a proptosis suggest fracture of the base of the skull.

2. The *pupils* are usually equal and of medium size in cerebral hæmorrhage. In hæmorrhage at the base, and in fracture of the skull, they are not uncommonly unequal, and one may react to light and the other not. Pin-point pupils are highly characteristic of opium poisoning, and of hæmorrhage into the pons.

3. The extreme infrequency of the *respiration* in opium poisoning is very characteristic. Respiration of the Cheyne-Stokes type or its modifications is found in apoplexy.

4. Unilateral rigidity or flaccidity of the *limbs* is often easily detected and indicates a gross cerebral lesion. In ventricular or pontine hæmorrhage there is either bilateral rigidity or bilateral resolution. The reflexes may vary on the two sides, and an extensor

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plantar response may be present on one or both.

5. *Optic neuritis* practically never occurs in simple vascular lesions. If present it usually indicates either tumour of the brain, syphilis of the brain, or Bright's disease. Rupture of an aneurysm at the base of the brain may be associated with optic neuritis, which is not unlikely to be unilateral.

6. The examination of the urine for albumin or sugar, and of the blood by Williamson's test for glucose, may show the presence of renal disease or diabetes.

7. The fluid obtained by lumbar puncture is always bloodstained in hæmorrhage within the dura mater, and hence this examination is of immediate service. In hæmorrhage from the middle meningeal artery no blood is found in the cerebro-spinal fluid; this may be of value in the diagnosis between intra- and extracerebral hæmorrhage. In thrombosis the fluid is clear.

Uremic coma.—The recognition of this condition may be difficult in the absence of other signs of renal disease; it is commonly broken by repeated convulsive attacks, the temperature is nearly always subnormal, and though hemiplegia may be found, it is certainly rare apart from coexisting gross lesions. A symptom in cerebral hæmorrhage which I have found of value is the constant difference in the temperature when taken in both axillæ; that on the paralysed side may be half to one degree higher than on the other.

Serous apoplexy.—In rare circumstances sudden effusions of serous fluid occur in the cranial cavity, and may rapidly cause coma simulating the apoplexy from cerebral hæmorrhage. In these cases there is rarely any indication of a hemiplegia, and resolution of the limbs is more likely to be present than rigidity. This condition may be met with in old people with impaired nutrition, after sunstroke, associated with infections of low virulence, after blows on the head, or even after emotional shock.

Acute hæmorrhagic encephalitis sometimes shows itself by early coma. The guiding features are a history of some preceding febrile disease such as influenza, headache and delirium, high fever, and usually the youthfulness of the patient. Apparently *hysteria* may resemble organic coma. One remarkable case is on record in which the coffin had actually been ordered. The foudroyant form of *cerebro-spinal meningitis* may be confused with cerebral hæmorrhage, especially if it be a sporadic case.

Ingravescent hæmorrhage with apparent recovery from the initial symptoms may be extremely difficult to diagnose with certainty, as the history of the following case shows. A man, *æt.* 60, while giving an address, suddenly lost the thread of his discourse; he did not lose consciousness but vomited several times, and complained of pain over the brows. He was collapsed, but able to walk home. Physical examination could detect no signs of disease; his heart and vascular system appeared normal. He gradually improved, but four days later suddenly became comatose and died. Lumbar puncture showed the presence of nearly pure blood within the theca.

An apoplectiform seizure due to embolism can be diagnosed only if a source for the embolus can be found. The differential diagnosis between cerebral hæmorrhage and thrombosis may be very difficult; syphilis may be the cause of either condition. Infective valvular diseases may cause either hæmorrhage or embolism.

A sudden hemiplegia in a previously healthy young man is most likely due to thrombosis secondary to syphilitic endarteritis; in a healthy young woman a thrombosis due to some primary disordered blood state is more probable. In children congenital syphilis may cause either hæmorrhage or thrombosis. The rare cases of granular kidney in children which may terminate by cerebral hæmorrhage are to be remembered.

The presence of renal disease in a person between 40 and 60 who has been the subject of an apoplectiform seizure is strongly in favour of hæmorrhage. Thrombotic lesions due to arterio-sclerosis occur at a later age. A strongly acting heart and a high blood-pressure are *pari-passu* evidence in favour of hæmorrhage, while cardiac feebleness and a depraved blood state are associated with a thrombosis. Repeated attacks of an apoplectiform nature are usually due to thrombosis. It must be admitted that in some cases a definite diagnosis cannot be made. In my experience the error has usually been in making the diagnosis of softening when the actual lesion was a hæmorrhage. Post-mortem findings, if taken alone, certainly show that thrombosis is a comparatively rare lesion.

Prognosis.—Cerebral hæmorrhage may be rapidly fatal. Of nearly a thousand patients with apoplexy admitted into a Vienna hospital, 57 per cent. died. The chief *unfavourable signs* are deep and persisting coma, a feeble rapid

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pulse, shallow respiration with an ashen grey pallor of the lips, evidence of rupture into a ventricle or of pontine hæmorrhage, ingravescent hæmorrhage with slowly developing coma, recurrent apoplectic seizures, and a rapid drop of the temperature to subnormal or its sudden rise to 103° to 105° F. *Favourable signs* are gradually lessening depth of coma, maintenance of a quiet, regular pulse of moderate force, and of a steady temperature about the normal.

The prognosis *quoad vitam* of cerebral embolism, except when due to infective endocarditis, is much better than in cerebral hæmorrhage. Of 273 cases collected by Ernest Jones (including embolism from infective endocarditis), 8 per cent. died within a day, 35 per cent. within a week, and 56 per cent. within a month. Prolonged and deep coma is of bad prognosis, but the main element in the outlook is the condition of the heart. Recurrences also are dangerous.

In cerebral thrombosis the prognosis is darkened by the fact that we are dealing with a progressive disease. Recurrences, multiple lesions and slowly progressing spread of the disease make the final outlook very bad. The prognosis of the attack itself is not so grave as in hæmorrhage; recovery may occur after a very long period of unconsciousness, though deep coma is of bad import. Thrombosis of the basilar artery is a very fatal condition. The prognosis of cerebral thrombosis due to syphilitic endarteritis is more favourable, as regards life at least, particularly in young and otherwise healthy subjects.

Reference may be made to the remarkable cases of recurrent attacks of coma associated with vascular disease of the brain; I have known such attacks to recur four times and be followed by complete recovery for a considerable period before the final fatal seizure. On each occasion the recovery was complete enough to permit the patient to return to his occupation. These cases are attributed to spasm of the vessels.

Treatment.—It is possible to do something to prevent the occurrence of a cerebral hæmorrhage if the tendency to it is recognized early enough. Such an opportunity occurs when the patient presents a high blood-pressure with signs of granular kidney, and perhaps has had transitory and apparently trivial cerebral disturbances. It is, however, to be borne in mind that many persons who die of cerebral hæmorrhage enjoy excellent general health and lead useful and busy lives. The restrictions

therefore imposed upon them should be wise and not too arbitrary. Attention is to be directed to controlling the output of work by the heart, moderating the blood-pressure, and favourably influencing body metabolism. In ordinary cases I do not consider it desirable to diminish largely the proteid intake by cutting off flesh meat, etc. Concentrated purin derivatives are best avoided, such as soups made from strong meat stock, beef tea, meat extracts, liver, kidney, and sweetbreads. Tea and coffee should be taken in moderation only. Some writers strongly object to allowing milk, cheese, or eggs. I am not able to subscribe to this prohibition, and advise a partial lacto-vegetarian dietary, and permit also cheese and eggs. The amount of liquid consumed should, however, be limited, since this throws more work on the heart. Warm, but not hot, baths several times a week are useful, and habitual gentle exercise in the fresh air is advisable.

With regard to drugs, 5 gr. of the mercury pill may be taken once or twice a week at bedtime, followed by a saline aperient in the morning; or if the bowels are constipated a saline draught may be taken every morning. Iodide of potash or iodine in organic combination is frequently given. Experience seems to show that it is useful. Iodides are best given in small doses for long times, separated by intervals of freedom. Sir Lauder Brunton advised the regular use of potass. bicarb. 30 gr., sod. nitrate 20 gr., and sod. nitrite $\frac{1}{2}$ gr. to 1 pint of water.

In the apoplectic seizure it is best that the patient should not be moved, though usually this is impossible. He should be laid in a quiet room, with the head slightly raised and the neck and thorax unimpeded by clothes. The bowels may be opened by placing 3 gr. of calomel or 1 min. of croton oil in a little sugar upon the tongue. If the patient can swallow, sips of water may be given, but food is unnecessary, and its administration may be dangerous if swallowing is affected. No attempt should be made to arouse him from coma. Stertor may often be diminished by placing the head sideways and drawing the tongue forward. It is often wise not to attempt active interference. The theoretical indications for the treatment of hæmorrhage and thrombosis are opposed, and the diagnosis cannot always be made. If the pulse be full and bounding in the early stage of the seizure and the patient not too old, venesection is a time-honoured

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remedy and has appeared to cut short the coma. It should certainly not be performed if he is weakly or if the heart is feeble. In this event thrombosis is possible and cardiac tonics, as digitalis and strychnine, may be given hypodermically. This treatment, combined with salines by the rectum, may be continued in cases of long coma (fourteen days or more) which run a quiet course and in which the fatal signs of a bulbar failure do not appear. When these vital centres obviously fail, treatment is useless. On the whole, it is unlikely that we can do very much to modify the outlook in cerebral hæmorrhage. Inactivity may be more serviceable than meddling therapy.

For the prognosis and treatment of syphilitic vascular disease of the brain, see CEREBRO-SPINAL SYPHILIS.

W. B. WARRINGTON.

CEREBRO-SPINAL FEVER (see MENINGITIS).

CEREBRO-SPINAL FLUID, PATHOLOGY OF (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

CEREBRO-SPINAL MENINGITIS (see MENINGITIS).

CEREBRO-SPINAL SYPHILIS.—The nervous system may be involved in any stage of syphilis. Examination of the cerebro-spinal fluid has shown that at least the meninges are affected in an early stage in a considerable number of cases, though there may be no clinical evidence of it; while in other patients nervous complications, as tabes dorsalis, may not develop till thirty or forty years after the original infection.

The nervous disease may also assume many forms. It may be a systemic degeneration of certain tracts, as in tabes; or the result of syphilitic vascular disease, as in cerebral or spinal thrombosis; or due to meningitis or gumma-formation; or to a combination of systemic and interstitial disease, as in general paralysis. Tabes dorsalis, general paralysis, and acute syphilitic myelitis are dealt with under TABES DORSALIS, GENERAL PARALYSIS OF THE INSANE, and MYELITIS; gummata are considered under CEREBRAL TUMOUR and SPINAL CORD, TUMOURS OF; and the effects of softening due to arterial disease, under CEREBRAL VASCULAR DISEASE.

Cerebro-spinal syphilis is a diffuse affection of specific origin which involves chiefly the

membranes, supporting tissues, and vessels, and in this way produces secondary lesions of the nervous tissue. The chief incidence of the affection may fall either on the brain or on the spinal cord, but in the majority of the cases both are involved. For the convenience of description they may first be considered separately.

Spinal Syphilis.—This may present itself in several different forms, but the most common is CHRONIC MENINGO-MYELITIS. Its chief **pathological lesions** are due to a gummatous infiltration of the soft membranes, which probably commences in the arachnoid; this and the pia mater become matted together and frequently attached to the dura mater by dense adhesions. Later the thickened meninges undergo fibrosis and contract, with the result that the spinal cord and its roots may be constricted. The vessels are simultaneously involved by periarteritis and endarteritis obliterans, and as it is chiefly the pial branches, supplying the periphery of the cord, that suffer, small foci of softening may develop here. The peripheral portions of the cord may be also damaged by an extension of the gummatous process along the penetrating vessels. The pathological changes are consequently found chiefly in the peripheral layers of the cord, but the whole transverse section may suffer when constricted. Acute myelitis is produced by a combination of softening and gummatous infiltration of one or more segments. Isolated or multiple gummata may grow in the meninges and produce the picture of a spinal tumour. In one important group of cases the disturbances are due to the collections of serous fluid in loculated spaces in the membranes, causing symptoms resembling those of a compressing tumour. All these changes may affect the greater portion of the cord, or may be limited to one region; in the latter case it is chiefly the middle and lower dorsal segments that suffer.

Symptomatology.—The disease generally begins with pains in the limbs or trunk, or a constricting girdle sensation, which are due to compression or invasion of the posterior roots. These are soon succeeded by a numbness, deadness, or coldness of the legs, associated with a progressive weakness, which generally takes the form of a spastic paresis. If, however, the anterior roots of the lumbosacral segments are affected, the muscles of the legs waste and the paresis remains flaccid. The gait is always of the spastic type, and is generally worse than would be expected from

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examination of the strength of the individual movements of the limbs. Sensation is, as a rule, not much disturbed, unless the spinal lesion is severe. The functions of the sphincters suffer early; there is most commonly a slowness and a difficulty in starting micturition, which may progress to actual retention of urine or to a dribbling incontinence. The knee- and ankle-jerks are much exaggerated, the plantar reflexes are of the extensor type, and the abdominal reflexes absent.

Severe involvement of one or more segments may produce the picture of a local transverse lesion, with a spastic paresis and loss of sensation below the corresponding level of the body.

When the disease affects the cervical enlargement and the anterior roots of this level are damaged, we have the condition of an atrophic palsy of the arms associated with spastic weakness of the legs. Not infrequently vascular or infiltrative lesions affect chiefly one side of the spinal cord only, and produce the Brown-Séquard syndrome, that is, motor weakness and loss of the sense of position on the side of the lesion, and loss of sensibility to pain and temperature on the opposite side. A similar lesion affecting the posterior columns produces loss of the sense of position in the legs and consequently an ataxic gait that may simulate that of tabes. This may be also a result of a partial lesion of the posterior roots of the lower segments—pseudo-tabes syphilitica.

Finally, spinal syphilis is often associated with cerebral symptoms and cranial nerve palsies, and especially with weakness of certain ocular muscles, ptosis, or pupillary abnormalities. The character of the disease depends on the multiplicity of its manifestations, their incompleteness, and variability, rather than on special clinical features.

Diagnosis.—The most important point in the diagnosis of spinal syphilis is its distinction from *disseminated sclerosis*. This is not always easy on clinical signs alone, but a history of syphilis, its predominance in males over the age of 35 years, the presence of sensory loss and pupillary signs, and the absence of nystagmus, intention tremor, and scanning speech, should always suggest a syphilitic disease. The presence of a positive Wassermann reaction in the blood is a valuable indication, but conclusive evidence is obtained only on examination of the cerebro-spinal fluid, in which there is always a large excess of cells and an increased

amount of albumin, and which frequently gives a positive Wassermann reaction.

ERB'S SYPHILITIC SPINAL PARAPLEGIA.—A special clinical type has been described by Erb. In most of these cases the pathological lesions are similar to those already described, but in others there is a combined degeneration of various columns of the cord which may be primary, or secondary to small myelitic foci. The characteristic symptoms are a slowly progressive paresis of the legs with a very spastic gait, though, to handling, the limbs may appear flaccid, or at least not so rigid as would be expected. Sphincter disturbances, especially retention of urine, develop early, pains are rare, and there is little or no loss of sensation. The knee- and ankle-jerks are very brisk, clonus can be obtained, and the plantar responses are extensor. In this type cerebral symptoms and cranial nerve palsies are uncommon. The disease is chronic, but it may become stationary, or temporary remissions may occur.

SYPHILITIC RADICULITIS.—It has been already pointed out that the spinal roots are frequently damaged by gummatous infiltration, or by constriction by the thickened and sclerotic membranes. Occasionally the cord escapes, and the only clinical signs are referable to these root lesions. When the anterior roots are affected we meet with weakness, wasting, and changes in the electrical reactions of the muscles supplied by the affected root or roots. This is always more prominent in the limbs, and particularly in the hands and arms, than in the trunk. It generally develops rapidly, and is unilateral or asymmetrical. The posterior roots are almost invariably affected at the same time, and radiating pains with sensory disturbances consequently occur in the area of their peripheral distribution. These pains persist for long periods unless the disease becomes sufficiently intense to destroy the sensory fibres. They are frequently excited by sudden movements of the spine; it is almost pathognomonic of posterior root irritation that coughing, and particularly sneezing, cause acute radiating pains (*signe d'éternuement*). Only one root may be affected, and then the symptoms are uniradicular; or several may be involved. The cauda equina is affected occasionally, and then a flaccid palsy of the lower limbs results, with severe pains which resemble those of sciatica, and sensory disturbance of root distribution.

CERVICAL PACHYMEINGITIS is frequently syphilitic. It is due to a massive thickening of

CEREBRO-SPINAL SYPHILIS

all the membranes over the cervical enlargement, which compresses the cord and involves the spinal roots at this level. There consequently result an atrophy of the hand and arm muscles and a spastic weakness of the legs, associated with local and distant disturbances of sensation, which may resemble that of syringomyelia. The distinguishing feature is the occurrence of violent radiating pains in the arms, which are rare in syringomyelia. This condition sets in subacutely, but after some weeks becomes stationary. It improves somewhat under energetic treatment.

SYPHILITIC MUSCULAR ATROPHY.—Apart from the atrophy produced by compression or involvement of the anterior roots by meningeal lesions, syphilis is occasionally the cause of isolated muscular atrophy. That which occurs in tabes may be due to this root disease, or to a selective action of the syphilitic virus on the motor cells or fibres. In another form the lesion is a progressive atrophy of the cells of the anterior horns of the cord, most commonly in the cervical region, which is probably secondary to an endarteritis of their nutrient vessels. It may be impossible to distinguish by clinical signs alone this condition from progressive muscular atrophy or chronic anterior poliomyelitis. If it is suspected, resource must therefore be had to examination of the cerebro-spinal fluid and blood by the Wassermann test. It is rare.

Cerebral Syphilis.—The pathological changes of cerebral syphilis resemble closely those that have been described in the section on spinal syphilis. There are diffuse, widespread, or local meningeal lesions of irregular intensity, which involve the underlying brain or cranial nerves in variable degrees; foci of softening due to vascular disease and gummata also occur. The last two conditions are dealt with elsewhere (see pp. 225, 218). The first form may conveniently be considered under *Vertical Meningo-encephalitis* and *Basal Gummatous Meningitis*.

VERTICAL MENINGO-ENCEPHALITIS is the less common form. It may develop in an early stage of syphilis. The first symptom is usually headache, which is often unilateral or local, and is associated with a local tenderness of the scalp or skull. One characteristic feature is its tendency to become worse at night. Vomiting and optic neuritis are rare, and unless the disease is extensive there are no pronounced mental changes. The local symptoms depend on the site of the lesion; when it

lies over the central cortex numbness, weakness, or paralysis of one or both of the opposite limbs, generally associated with local epileptic spasms or convulsions, is the most prominent symptom. In addition to local Jacksonian attacks, generalized seizures, indistinguishable from ordinary epilepsy, may occur. The variability of the symptoms, their tendency to progress by a series of steps rather than uniformly, and their remission under treatment are suggestive of their cause.

BASAL GUMMATOUS MENINGITIS generally begins in the arachnoid cistern of the interpeduncular space, and may spread from here forwards beyond the chiasma, or backwards over the ventral surface of the brain-stem. The cranial nerves are consequently involved in a dense gummatous or fibrous thickening, which may also block the circulation of the cerebro-spinal fluid and produce hydrocephalus. Headaches, vomiting, giddiness, and optic neuritis, the general symptoms of increase of intracranial pressure, are usually prominent. Attacks of stupor or unconsciousness are not uncommon, or the patient may be dull, drowsy, irritable, and amnesic, or may pass into a comatose state during the acute stage. The local symptoms, which are due to palsies of the cranial nerves, are more characteristic. The third or oculo-motor is the nerve which is most liable to suffer, and squint, diplopia, ptosis, and irregular and uneven pupils which are often inactive to light, consequently develop. One or both nerves may be affected. The other ocular nerves, the fourth and the sixth, are less frequently involved. When one trigeminal nerve is affected the symptoms commence with neuralgic pains in the same side of the face; this is followed by anæsthesia, weakness of the masticatory muscles, and often keratitis. The facial and auditory nerves are less frequently paralysed, but unilateral palsy of the palate, larynx, and tongue is often seen. These cranial nerve palsies may be complicated by the presence of symptoms due to gummatous or vascular lesions of the brain-stem. The optic nerves, chiasma, or tracts may also be compressed or invaded, so that the patient complains of dimness or loss of sight, and on examination unilateral amblyopia, hemianopia, and more commonly central or paracentral scotomata, are discovered. Symptoms of diabetes insipidus (polydipsia and polyuria) have been frequently recorded in cases of basal meningitis; they are probably due to involvement of the pituitary gland.

CEREBRO-SPINAL SYPHILIS

Diagnosis of cerebral syphilis.— Frequently the diagnosis is not easy, as the symptoms are variable, inconstant, and not characteristic. The age of the patient, a history or probability of previous infection, or other evidences of such, should always raise a suspicion, and it is then the duty of the doctor to avail himself of whatever aid the pathologist can offer him. A positive Wassermann reaction in the blood can only support this suspicion; it is by the examination of the cerebro-spinal fluid that it can be confirmed or refuted. It should be remembered that in meningo-vascular syphilis an excess of cells and an increase of albumin are more constant than a positive Wassermann reaction in the fluid.

Treatment.— Cerebro-spinal syphilis demands as early and as vigorous treatment as is possible. Mercury is unquestionably the most effective drug, and, if circumstances permit, is best administered by inunction. This should be commenced by the vigorous rubbing in of one drachm of ung. hydrarg. twice a day for the first week or ten days; then the dose may be reduced to one drachm a day and continued till salivation occurs, or until the patient notices the metallic taste. There is little danger of severe stomatitis if the patient uses his toothbrush regularly; an astringent mouth-wash is also advisable. The inunction should be made in different regions on successive days, as in the axillæ, front of the chest, groins and inside of the thighs, and it is more effective if the patient has a hot bath, or is washed thoroughly with very hot water and soap, before each application. As soon as signs of stomatitis develop, the drug should be stopped for ten to fourteen days and the patient put on a tonic or an iodide mixture. A second or third course is frequently necessary. The administration of mercury per os should be continued long after the acute stage is over.

The organic arsenical preparations are also valuable, but should not replace mercury. Novarsenobillon has proved very safe and effective; a series of six or more intravenous injections, starting with 0.3 grm. and increasing to 0.9 grm., may be combined with mercurial treatment. Salvarsan or neo-salvarsan may be employed instead; galyol is less satisfactory. Iodides, which are so popular in the treatment of late syphilis, are less useful when the nervous system is involved, except, perhaps, when the lesions are mainly vascular. Then potassium

CERVICAL RIB

iodide in doses of 30–60 gr. a day, or iodipin, may be combined with mercury or alternated with it.

Finally, it must be remembered that as large gummata consist chiefly of necrotic tissue, they can be little influenced by drugs, and that consequently when they produce local symptoms their surgical removal must be considered.

W. B. WARRINGTON.

CERVICAL ADENITIS (*see* LYMPHATIC GLANDS, ENLARGEMENT OF).

CERVICAL ENDOMETRITIS (*see* ENDOMETRITIS, ENDOCERVICITIS, AND METRITIS).

CERVICAL EROSION (*see* ENDOMETRITIS, ENDOCERVICITIS, AND METRITIS).

CERVICAL PLEXUS, LESIONS OF (*see* SPINAL NERVES, LESIONS OF).

CERVICAL RIB.—In a certain number of people a rib articulating with the seventh cervical vertebra may be developed on one side or on both sides, but its presence is not necessarily followed by nervous phenomena. When such symptoms develop they take the form of pain, usually referred down the inside of the arm to the wrist or little finger, followed later by muscular wasting of the hand and anæsthesia of a strip along the inside of the forearm. Vaso-motor phenomena are also sometimes very pronounced; cyanosis, pallor, œdema, and even gangrene of the fingers have all been met with. Cervical ribs vary greatly in length and are usually much curved, and can be felt as a hard swelling in the supra-clavicular fossa; they may be attached to the first rib by a true costal cartilage, or this may be absent and the tip of the rib may be continued by a firm fibrous ligament to be attached to the first rib. It will be remembered that normally the first dorsal nerve bends over the neck of the first rib to join the eighth cervical nerve and form the inner cord, and this arching of the lowest large nerve entering the brachial plexus over the rib may sometimes lead to injury of that nerve in cases of violent wrenches of the arm in a downward direction; a form of paralysis known as Klumpke's palsy, in which either the first dorsal nerve or the whole inner cord may be injured, is produced in this way. When a cervical rib is present, the first dorsal nerve, in order to reach the plexus, has a much more arched course, as it rises not only over the first rib but over the cervical rib, and it is consequently liable to injury from pressure

of this accessory rib. Not only may the first dorsal and eighth cervical nerves thus be injured by pressure, but the subclavian artery and vein may suffer in the same way, giving rise to oedema, cyanosis, diminution of the radial pulse, and gangrene of the fingers, as already mentioned. Strong pulsation of the subclavian artery in the neck may also be felt.

Symptomatology.—Symptoms rarely, if ever, develop until after puberty; they are usually found between the ages of 16 and 28. Either sex may be affected, women, however, much more often than men. The reason put forward to explain this age and sex incidence is that the shoulders are much squarer in children and in men than in girls after the age of puberty; the sloping shoulder necessitates greater arching of the first dorsal nerve and subclavian artery on their way into the neck.

The sensory phenomena produced by the pressure of the cervical rib upon the first dorsal nerve and the inner cord are usually the most prominent symptoms. These consist of paræsthesiæ, of tinglings and numbness, often amounting to pain, referred to the inner border of the forearm and sometimes to the little and ring fingers. The pain varies in severity and in the times of its occurrence, being usually worse when the arm is pressed downwards as in any lifting effort, or in carrying a heavy bag. After a time, alterations in the sensibility of the first dorsal root area on the inside of the forearm may be demonstrated by loss of appreciation of light touch and diminution in the apparent sharpness of a prick. This partial anæsthesia may spread to the little and ring fingers, but sensation to pressure is never lost. Atrophy of the interossei and other intrinsic muscles of the hand, including those of the thenar eminence, accompanies or may even precede the development of the ulnar anæsthesia, giving rise to a clawed appearance of the hand. As stated above, vaso-motor phenomena may accompany the sensory symptoms, leading to oedema, pallor, or cyanosis of the fingers. The development of the muscular atrophy is usually very slow and insidious, so that reaction of degeneration is not found. This condition is occasionally bilateral; it may then be mistaken for progressive muscular dystrophy, which, however, can be excluded by the sensory phenomena.

Treatment.—This practically resolves itself into the question of the advisability of surgical interference. In the slighter cases of paræ-

thesiæ massage of the limb may be tried, and the patient advised to avoid keeping the arm hanging down and not to carry heavy weights. In all cases in which the presence of cervical rib is suggested by the symptoms, even if no bony prominence can be felt in the neck, an X-ray photograph of the neck should be taken. Bilateral cervical ribs are often found to be present when the symptoms were referred entirely to one side only. When the pain is almost constantly distressing, or if gangrene of the fingers is threatened, or the other vaso-motor symptoms are very troublesome, then it may be considered advisable to attempt an operation for the removal of the cervical rib, after its presence has been fully demonstrated by a skiagram. The operation is by no means an easy one, and it may sometimes be impossible to relieve the cause of pressure upon the involved structures, but quite a number of cases have been treated very successfully by this method. In certain cases vague pains and paræsthesiæ about the shoulder and arm, not specially along the course of the inner cord of the plexus, sometimes persist after an apparently successful removal of the rib. In some cases the characteristic symptoms may be due to stretching of the first dorsal nerve over the first rib, no cervical rib being present. Relief may be obtained by excising part of the first rib. Such symptoms may have been of a functional nature throughout; it is said to be not uncommon to find the symptoms of neurosis associated with the congenital deformity of an extra cervical rib.

WILFRED HARRIS.

CERVICITIS (*see* GONORRHOEA; ENDOMETRITIS, ENDOCERVICITIS, AND METRITIS).

CHALAZION (*see* EYELIDS, AFFECTIONS OF).

CHANCROID (*see* ULCUS MOLLE).

CHARBON (*see* ANTHRAX).

CHARCOT'S ARTHROPATHY (*see* TABES DORSALIS; SYRINGOMYELIA).

CHARCOT - MARIE - TOOTH DISEASE (*see* MUSCULAR ATROPHY, PERONEAL).

CHEIROPOMPHOLYX (*see* POMPHOLYX).

CHELOID (Gr. *χηλῆ*, a claw). A benign fibrous tissue tumour which tends to recur locally after excision.

CHELOID

Etiology.—Opinions on the question of causation are divided, and the only established fact at the present time is that the development of the condition always proceeds in the scars of accidental traumata or of operations. The exciting agent may be of a microbic nature. Darier, Tilbury Fox, and others regard cheloids as tuberculides or the manifestation of a tuberculous tendency. Others, again, believe that the "terrain" is all-important, and that the patients have developed or inherited a peculiar reactivity of scar tissue.

Histopathology.—Repeated microscopic examination has never revealed anything more than massive, sharply defined strands of fibrous tissue arranged in elongated bundles and running parallel to the skin surface. They are situated invariably in the subepidermal connective-tissue layers, i.e. in the dermis itself. The papillæ and epidermis are normal, which is not so in sections of hypertrophic scar tissue.

Symptomatology. The growth of the cheloid is usually a slow and insidious affair, requiring from two to three months to as many years to reach maturity. Rapid growth does, however, sometimes occur: in one case full development was attained in six weeks after the patient had left the hospital with a healed scar. Of firm and leathery texture, and usually elongated, in correspondence with its microscopic arrangement, the cheloid is a sharply defined, smooth, prominently raised, pink to ivory-white tumour of irregular arborescent shape, with frequently a glistening surface due to the stretched but otherwise normal epidermis. All cheloids have the following tendencies in common: (1) Recurrence *in situ* on removal. (2) Invasion of normal, unscarred skin in the immediate neighbourhood of the original lesion. (3) Ulceration or malignant degeneration if chronically irritated. The subjective symptoms usually associated with development of the condition are burning and pricking pains, worse in wet weather and at night time. A cheloid may either persist indefinitely or may ultimately, after many years, undergo gradual involution and disappear completely, leaving behind it only the original scar.

Diagnosis.—The appearance, history, and course of the lesions conform so closely to type in every case that mistakes in diagnosis must be very rare. The possibility of confusion with hypertrophy of scar tissue can be guarded against by remembering that cheloidal changes are not, as a rule, confined to the scar itself but tend to invade the surrounding skin.

CHEST, DEFORMITIES OF

Treatment.—Formerly the most hopeless of all benign cutaneous growths from the point of view of treatment, the condition may with considerable confidence be expected to yield to properly adjusted and filtered doses of the Röntgen rays and to radium. I have no practical experience of the latter method, but I am courteously informed by the authorities of the Radium Institute that, while the risk of ulceration and telangiectases after repeated doses must not be ignored, on the whole the treatment is satisfactory. (See RADIUM-THERAPY.) The results with X-rays are equally good. The rays are passed through aluminium plates 0.5 mm. in thickness, and weekly exposures of $\frac{1}{2}$ S.N. are given until 1½–4 full pastille doses have been administered. The surrounding skin must always be most carefully shielded with lead-foil. Several months should be allowed to elapse before a fresh course of the rays is given, and in the interim German authors recommend stimulation by the Kromayer lamp or scarification, which is said to sensitize the tumour to fresh applications. After the second or third exposure the subjective symptoms usually disappear, and in from three to six months very sensible diminution in the size and prominence of the lesion becomes apparent. Besides the radiotherapeutic treatment, none other is worthy of consideration. HENRY SEMON.

CHEMICAL PATHOLOGY (see PATHOLOGY, CHEMICAL, MODERN DEVELOPMENTS OF).

CHEMOSIS (see CONJUNCTIVITIS).

CHEST, DEFORMITIES OF.—Abnormally shaped chests may for convenience be divided into (1) those in which the chest is affected generally, (2) local abnormalities.

1. ABNORMALITIES OF THE CHEST AS A WHOLE

(a) **The flat chest.**—The costal cartilages are devoid of their usual convexity forwards and run in a more or less straight line to their sternal junctions. In consequence the distance between the sternum and the vertebral column is lessened. Expansion of the chest is therefore imperfect.

(b) **The alar chest.**—In this the scapulae are "winged," their vertebral borders and inferior angles projecting unduly; and the shoulders slope more than normally, the neck appearing long and the larynx prominent. The ribs incline obliquely downwards, and hence the chest is somewhat flattened and

CHEST, DEFORMITIES OF

diminished in capacity though longer than normal.

Not infrequently these two varieties are combined, and in themselves do not imply the presence of either past or active disease. They are hereditary and are found especially in families who are predisposed to tuberculosis; they have been called "phthisoid" chests.

(c) **The rachitic chest.**—The ribs in rickets, being soft, may yield as the result of atmospheric pressure, and do so at their weakest part, the costo-chondral junctions. Hence, a groove is formed in this position running downwards and forwards in a line with the costo-chondral junctions. The lateral aspects of the chest have the appearance of being scooped out. At the bottom of the groove the "rickety rosary" may be felt. The lower costal margin may be everted, allowing the liver and spleen to be felt with unusual ease, and thus giving the impression that these organs are enlarged. Harrison's sulcus is often seen: this is a horizontal groove running around the lower part of the chest just above its everted margin. It may often be traced as far outwards as the midaxillæ. In severe cases the lateral constriction may be so great that the chest is roughly divided into two compartments, the anterior being the smaller; and a transverse section is somewhat fiddle-shaped. These features are accentuated if there be naso-pharyngeal obstruction.

(d) **The pigeon breast** may be congenital or may result from mouth-breathing. The lateral convexity of the ribs is less than normal, the ribs on the two sides inclining sharply from their angles to the sternum, which is thus thrust prominently forwards and projects beyond the plane of the abdominal wall, a sharp dip being produced at its lower end. The shape of a transverse section of such a chest is roughly triangular.

(e) **The barrel-shaped chest** occurs in emphysema. The lateral convexities of the ribs are more marked than normal, and they slope downwards to a less degree, taking a more horizontal position. The chest may be said to be fixed in the position of full expansion, and inspiration is assisted by the accessory muscles, the chest being elevated as a whole. Its sides are rounded and its depth increased. The clavicles are bowed forwards, and the sternum is more arched than normal. Emphysema of the apices of the lungs produces a fullness above the clavicles. This, together with the elevation of the chest, and a slight

projection forwards of the head which is often present, gives the patient the appearance of having a very short neck. In kyphosis a somewhat similarly shaped chest may be found.

2. ABNORMALITIES AFFECTING PART OF THE CHEST

(a) **Altered shape of one entire side of the chest.**—*Unilateral enlargement* occurs when the pleural cavity contains a large amount of free fluid, whether serous, hæmorrhagic, or purulent, and in pneumothorax. Large neoplasms of the lung or pleura may have the same effect, as may also compensatory hypertrophy of the lung when that of the opposite side is largely rendered inactive, as in fibrosis. *Unilateral shrinking* of the chest occurs when the corresponding lung is incapable of expansion or diminished in size. It is found, therefore, in fibrosis of the lung, whether tuberculous or produced in other ways, in collapse or after compression, and in cases of thickened and adherent pleura. The larynx and trachea are usually displaced towards the same side in the case of a shrunken chest, and towards the opposite when one side of the chest is enlarged.

(b) **Local abnormalities in shape.**—*Local bulging* of the intercostal spaces occurs in effusion and pneumothorax. Although this may be accompanied by some bulging of the ribs, the latter are more commonly pushed out by solid tumours. Undue prominence of the præcordium occurs in hypertrophy of the heart and in pericardial effusion; in the latter the intercostal spaces in this region are relatively more prominent. On the right side tumours, cysts, or abscesses of the liver may produce some prominence of the lower part of the chest, whilst subphrenic abscess occasionally has the same effect on the side in which it is located. Emphysema of the apices of the lungs produces fullness above the clavicles. *Local shrinking* is often noticed at the apices, the result of phthisis, definite hollows being produced above and below the clavicles. This is due chiefly to the associated fibrosis, and is often marked when excavation is present. Recession of the intercostal spaces occurs over fibrosis of lung and pleural adhesions. The *funnel-chest* (*Trichterbrüst*) is another form of local recession. A funnel-shaped depression is found in the lower part of the middle line of the front of the thorax. It may be congenital, or result from naso-pharyngeal obstruction or chronic



PLATE 7.—CHICKENPOX, WITH ABUNDANT RASH.

(Ricketts and Byles.)

CHEYNE-STOKES RESPIRATION

obstruction of the upper air-passages. Sometimes it is an occupation deformity, as in boot-makers and carpenters, and produced by mechanical pressure.

For alterations in shape due to curvature of the spine, *see* SPINAL CURVATURE.

FREDERICK LANGMEAD.

CHEYNE-STOKES RESPIRATION.—

In this condition the respirations occur in groups which are separated from each other by periods of apnoea. In each respiratory cycle the respirations are at first shallow, but gradually deepen until an acme is reached, when they as gradually lessen again. In this, the dyspnoic stage, about 8 to 30 respirations may occur. The duration of the apnoea is variable, and may last as long as 40 seconds.

This periodic respiration may be seen in normal infants during sleep, and is met with in healthy old age. It may be induced by rarefied air as on high mountains or during air navigation. It has been noted after poisoning by narcotic drugs. The common clinical causes are heart disease, especially in myocardial degeneration, arterio-sclerosis, uræmia, meningitis, depressed fracture of the skull, apoplexy and other forms of cerebral compression, and sunstroke. There appears to be an hereditary predisposition to Cheyne-Stokes breathing.

Usually, the blood-pressure and the pulse-rate lessen during the dyspnoic phase, but the opposite may be true in the cases associated with cerebral compression, both increasing with the deepening of the respirations. Towards the end of the dyspnoea there may be forced movements and an increase of muscular power. The pupils may dilate during the apnoic phase and return to normal size during the period of respirations. It has been shown that the alkalinity of the blood is diminished and the carbon dioxide in the alveolar air increased at the beginning of the dyspnoea, at which time also cyanosis is most in evidence.

Cheyne-Stokes respiration was formerly regarded as a herald of approaching death, but so many exceptions have been recorded that it is unwise to base a definite prognosis on its presence alone. It was noted by one observer throughout a period of sixteen years in the case of an elderly man who died at the age of 92.

Related to this form of respiration are two other varieties, (1) that described by Biot, and (2) simple grouped respirations. These

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are more commonly seen in children than is true Cheyne-Stokes breathing, and are especially met with in tuberculous or diplococcal meningitis. In Biot's form the respirations during the dyspnoic phase gradually deepen and then cease, and do not wane again as in the Cheyne-Stokes breathing. In the simple grouped form, respirations occur in groups of two, three, or more breaths, or in alternations of these.

FREDERICK LANGMEAD.

CHICKENPOX (*syn.* Varicella).—An acute febrile contagious disease characterized by a vesicular eruption, which usually appears in successive crops.

Etiology.—Like variola, varicella is not confined to any particular climate but is universally prevalent. The great majority of cases occur in children, especially between the second and seventh year, but the disease is not very uncommon among adults, in whom it is particularly liable to be mistaken for smallpox, especially when that disease is epidemic. One attack usually confers permanent immunity, but instances of a second and even a third attack are on record. The causal organism has not yet been isolated. The contagion is usually conveyed directly from the sick to the healthy, but it may be transmitted by a healthy third person or by fomites. The disease has occasionally been inoculated, prophylactic vaccination with lymph taken from the vesicles producing sometimes a localized or more rarely a generalized eruption.

Isolation and quarantine.—The patient should be isolated until all the scabs have separated. The quarantine for contacts is twenty-one days.

Symptomatology.—The incubation period is usually 14 days, but may range from 11 to 23 days. It is rarely shorter, however, than 12 or longer than 19 days.

In children the rash is usually the first manifestation. In some cases, however, especially in adults, the eruption may be preceded for one or two days by a varying degree of headache and backache, sometimes as severe as in the prodromal stage of smallpox. The eruption (Plate 7) nearly always appears first on the trunk, where it is most copious, then on the face and scalp, and last of all on the limbs, where the lesions are usually sparse, especially towards their distal extremities. The mucous membranes of the mouth, throat, and vulva are frequently affected, but the conjunctiva and cornea usually escape. As a

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rule, when the eruption is first seen the majority of the lesions are already vesicular, but a certain number do not progress beyond the papular stage. The vesicles, many of which are surrounded by a reddish areola, are oval or circular in shape, and contain a clear yellow fluid which in the course of one or two days becomes turbid and straw-coloured, when the vesicle begins to desiccate. Finally, a crust is formed which on separating leaves a pigmented mark on the skin. With rare exceptions the vesicles are not umbilicated, and are usually unilocular. Fresh crops continue to appear for a period ranging from three to ten days, according to the severity of the case.

The temperature, as a rule, does not exceed 102° , and often does not rise above 99° F., or is even normal, and is usually not raised for more than two or three days. In rare instances, as in a case of confluent varicella reported by the writer, the primary fever lasts a week, ranging from 103° to 104.8° F., and may be succeeded by a secondary fever due to the formation of numerous areas of suppuration beneath the scabs.

The eruption often tends to be confluent in areas where the skin has been previously irritated, e.g. by blisters or by skin eruption, such as eczema. In infants the rash is sometimes more profuse on the lower limbs than on the trunk, owing to the irritation caused by the fæces and urine. The eruption is often unusually abundant after a recent attack of scarlet fever.

Accidental rashes may develop at any stage of the disease, but prodromal and concomitant ones are much commoner than the post-eruptive. In order of frequency they are scarlatiniform, purpuric, morbilliform, and mixed. The trunk is most frequently affected, but there is no site of predilection similar to the abdomino-femoral region in the prodromal rashes of smallpox. The accidental rashes of varicella, like the prodromal rashes of measles, are usually of short duration, and are neither accompanied by cutaneous irritation nor followed by desquamation. From a diagnostic standpoint these rashes are of considerable interest, especially as the prodromal scarlatiniform rash is likely to lead to a mistaken diagnosis of scarlet fever. On the other hand, unlike the prodromal rashes of smallpox, they do not possess much prognostic value. Unless accompanied by hæmorrhages from the mucous membranes, the occurrence of purpura does not justify a grave prognosis. The rashes are

probably septic or toxic in nature, just as are the accidental rashes of the other acute exanthemata, and are independent of the varicella infection itself. Their greater frequency in childhood is to be explained not only by the predilection of varicella for young persons, but also by the greater sensitiveness of the skin and more active capillary circulation in the child.

Association with other diseases.—Varicella and scarlet fever not uncommonly concur. Between 1899 and 1907, 709 cases, or an annual average of 78.7 cases, in which the two diseases were concurrent, were admitted to the Metropolitan Asylums Board hospitals. Pre-existent or coincident scarlatina appears to exercise an unfavourable influence on varicella, a relatively high percentage of accidental rashes, gangrene, and confluent attacks occurring when the two diseases are associated. Varicella is not infrequently met with in association with other infectious diseases, such as measles, diphtheria and smallpox.

The possibility of the identity of herpes zoster and varicella has recently been suggested by several writers on the following grounds: (1) Chickenpox in one individual has followed herpes zoster in another within the ordinary incubation period for the former disease, when no other source of infection except herpes zoster was discoverable. (2) Much less frequently herpes zoster has apparently been contracted from a case of chickenpox. (3) The two conditions may sometimes co-exist. The subject needs further investigation.

Complications.—The most frequent complications of varicella are due to secondary infection of the pocks, which gives rise to various degrees of dermatitis, such as boils, subcutaneous abscesses, erysipelas, and impetigo. The most serious condition is *varicella gangrænosa*, in which one or more of the pocks undergo necrosis. It is most likely to occur in ill-nourished children debilitated by some pre-existing disease, especially by one of the acute exanthemata or by tuberculosis. Its gravity is shown by the fact that of 43 cases collected by Kiefer, 29 proved fatal. Varicella gangrænosa is sometimes associated with hæmorrhagic varicella, two forms of which have been distinguished—(1) a mild form in which the hæmorrhages are confined to the eruption, (2) a grave form characterized by petechiæ, ecchymoses, and hæmorrhages from the mucous membrane.

Nephritis, which Henoch was the first to

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describe as a complication of varicella, is most likely to occur in the first or second week of the disease. Although in most cases it runs a mild course, it occasionally proves fatal.

Ocular complications.—A slight degree of ocular involvement is not uncommon. In a series of 150 cases of varicella observed by the writer the eyes were affected in 13, or 8.6 per cent. Of 10 in which there was catarrhal conjunctivitis, there was palpebral œdema in 4, and considerable chemosis in 2, while 2 showed blepharitis, and 1 palpebral gangrene which healed, leaving only a very slight degree of ectropion. Serious ocular complications such as keratitis, corneal ulcer, iritis, panophthalmitis, ocular palsies, and optic neuritis, which have been recorded by various writers, are extremely rare.

Aural complications, according to Jacod, are much more frequent than is supposed. Early otitis due to spread of the bucco-pharyngeal inflammation along the Eustachian tube must be distinguished from the late otitis which is an aural localization of the general infection, and is more serious and of longer duration than the earlier variety. As a rule, the aural complications of varicella are milder than those of scarlet fever or diphtheria, but grave complications may arise, such as mastoiditis, meningitis, cerebral and cerebellar abscess, and thrombosis of the lateral sinus.

Nervous complications are extremely uncommon. Isolated cases of encephalitis, neuromyositis, peripheral neuritis, and poliomyelitis have occurred during or after varicella, but their etiological relationship is extremely doubtful, and concurrent infections, especially latent syphilis or diphtheria, must be excluded.

Laryngitis occasionally complicates varicella. It appears shortly before or soon after the eruption, and may be sufficiently severe to necessitate intubation or tracheotomy. Anatomically it is characterized by small circular ulcers situated generally on the vocal cords, the surrounding mucosa being more or less hyperæmic (Marfan).

The rarer complications of varicella include pleurisy, bronchitis, broncho-pneumonia, synovitis, arthritis, thyroiditis, and parotitis.

Diagnosis.—Apart from smallpox (q.v.) the disease most likely to be confounded with chickenpox is *impetigo*. The differential diagnosis is difficult only when old scabbing lesions are scattered about the trunk and proximal ends of the limbs. In such cases a distinction may be impossible if the eruption has not been seen

CHILBLAIN

in the early stage. Lesions in the mouth and throat, which are not affected by *impetigo*, are in favour of varicella.

Prognosis.—More than a hundred years elapsed after varicella was differentiated from variola by Heberden in 1767 before the prognosis of chickenpox was regarded as otherwise than entirely favourable. The recognition, however, of nephritis as an occasionally fatal complication, of varicella gangrenosa, and later still of hæmorrhagic forms of chickenpox, has tended to modify the teaching of Trousseau that no physician had ever seen a patient die of chickenpox.

Treatment.—Since it is impossible to foretell the future extent of an eruption, the prophylactic use of boric-acid baths, 1 oz. of the crystals to the gallon of water, morning and evening, is to be recommended. The cutaneous irritation which is often so troublesome in the disease is thereby considerably relieved, and the tendency to scratch is checked so that the amount of subsequent dermatitis is reduced to a minimum. In some children cardboard splints for the arms or gloves may be required, in addition to keeping the nails short and clean.

The patient should be given a low diet during the febrile stage, and should be kept in bed until the lesions have desiccated. The urine should be examined frequently throughout the attack owing to the occasional occurrence of nephritis. In varicella gangrenosa an attempt should be made to improve the general condition, and the lesions should be treated by prolonged soaking in a hot bath, followed by application of lotio hydrarg. perchlor. (1 in 2,000). J. D. ROLLESTON.

CHILBLAIN (*syn.* Erythema Pernio).

Etiology.—This common and often troublesome affection of the skin is due to the effect of cold or of rapid lowering of the temperature on the tissues and the blood-circulation of the affected parts. It may be regarded as a mild manifestation of frostbite (q.v.). Chilblain occurs in both sexes and at all ages, but is more common in the young and in persons whose health is not robust. This defect of health is frequently associated with chronic infections of various kinds, of which tuberculosis is one. Children liable to chilblain not infrequently suffer from chronic tuberculous infections of the lymphatic glands and other tissues. The disease is of special medical interest on account of the relationship of some of its phenomena with

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certain conditions depending apparently upon either physiological or pathological defect in the control of the circulation. Of these the condition known as acroasphyxia is one; mild examples of it have been described as the "chilblain circulation" on account of the fact that the affected skin closely resembles the early stages of severe chilblain. In such conditions the balance of the circulation may be disturbed in other respects than the stagnating erythema of the extremities. For instance, although many of these patients have a normal pulse-rate, in certain cases there is well-marked bradycardia, the pulse-rate being reduced to 50 or even fewer beats a minute, while in other cases the pulse-frequency may mount to 150-200. It is worthy of note, however, that some of these cases, which may be described as stagnation-erythema, are not distinctly characterized by chilblains, even when the hands, feet, nose, face, and ears are unusually livid. As chilblain is usually a definite skin trauma due to cold, it occurs occasionally in adults who are perfectly healthy and robust. Examples are met with in such persons who are accidentally exposed to cold and damp. For instance, if such a person moves from a warm, dry house to one situated on clay, badly built and damp, chilblains may appear for the first time on the approach of winter. The relationship of such chilblains to the severe circulatory disturbance made familiar during the War, and known as "trench foot," is of interest.

Pathology and symptomatology.—The *first stage* of chilblain, probably in all cases, is arrest of the circulation in the exposed or affected area owing to contraction of the blood-vessels. This stage of blanching may be very transient, but sometimes the condition of "dead fingers" may occur. Following on this is the *second stage*, a congestion of the part owing to distension of the capillaries; the characteristic reddish or purple chilblain is then noted. Succeeding this is the *third stage*, perhaps associated with more active hyperæmia, producing the sensation of heat and itching which is so familiar to the victim. These stages of dilatation or, it may be, temporary paralysis of the circulation may last for a long time; usually, however, with a higher temperature the vascular control is rapidly regained and the chilblain gradually disappears. During the stages of stagnatory erythema and of hyperæmia, blood-serum transudes to some extent into the connective-tissue spaces, producing definite swelling and occasional vesication.

The occurrence of itching entices the patient to rub or scratch the affected part, with the result that the sodden epithelium is removed, and the *fourth stage* of the chilblain, viz. an abraded or ulcerated surface, supervenes. The skin is now liable to the accidents of bacterial infection, and the ulceration may be slight or severe according to the nature of the infection and the patient's power of resistance. The ulcers are sometimes very slow in healing. In addition to the effects on the surface, the deeper tissues may become involved, with the result that thickening of the connective tissues round the joints of the fingers and the toes is not uncommon, and a condition resembling some forms of osteo-arthritis may occur. A certain thickening of the tissues so produced may remain permanently.

The parts of the body usually affected are the hands, the feet, the ears, the face, and the nose—areas usually exposed and in which the condition of stagnation-erythema most commonly occurs. In mild cases one or two chilblains only may occur, but in more severe cases the greater part of the surface of the hands may be affected with extensive ulceration, producing inability to work; similarly the feet may be so affected that walking for the time is impeded or impossible, while the occurrence of a chilblain on the tip of the nose is not only a great disfigurement but is often very difficult to heal.

Treatment.—Preventive treatment is of the first importance. Those liable to chilblain should seek to maintain as high a standard of health as possible, by sufficient or even vigorous exercise, especially in cold weather. A short course of gymnastic exercises for the arms and hands on rising in the morning, and a sharp walk after breakfast, are to be recommended. Persons subject to chilblain are likely to suffer if their occupation is sedentary, and especially if they happen to live on damp clay soils, or in badly built, damp houses. The body and extremities should be clothed lightly but warmly; any constriction or compression of the extremities by tight garments is likely to induce chilblain. The more usually affected parts, such as the hands, should be kept as equally warm as possible by means of gloves, or in other ways. Dressed leather gloves should not be worn in the winter. The food, especially in the case of delicate children, should be so chosen as to maintain a good state of nutrition. For purposes of ablution warm baths should be used, and the skin made to

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react afterwards by well-applied friction. The parts especially liable, such as the hands and feet, should always be washed in warm water, and care taken to prevent the reaction of cold subsequently. There is frequently a tendency to hyperidrosis in persons showing stagnation-erythema. Special precautions should be taken to keep the hands and feet both dry and clean. A mildly antiseptic dusting powder, applied after thoroughly drying the skin, is of much service—e.g. boric acid 15 per cent., zinc oxide 15 per cent., finely powdered siliceous earth (talc or cimolite) 70 per cent. A small quantity of camphor or menthol added to such a dusting powder is often useful and grateful to the patient.

During the first stage of the chilblain the hands may carefully and gradually be warmed, and the circulation stimulated by friction or by exercise. In the second stage, that of capillary dilatation, it is well not to be too vigorous in treatment; the hands should be kept at an equable warmth, and patience exercised till the vascular tone is re-established. In this and the hyperæmic stage, characterized so frequently by itching, the patient injures the surface by scratching or rubbing. It is in these stages that the numerous chilblain lotions and ointments sold in such quantities during the winter are found to be comforting. Such applications contain, as a rule, antipruritics or slight counterirritants, such as turpentine, menthol, camphor, iodine, carbolic acid, and alcohol, in various strengths and various forms. They often produce a feeling of comfort on application, but they should be used with care, as they are all likely to damage or even macerate off the soaked epidermis. A well-known example of such an application is tinct. cantharidis 2 dr., lin. terebinthinæ acetatum 4 dr., lin. camphoræ 2 fl. oz.

The ulceration which, in spite of the care taken by the patient, not infrequently occurs is often difficult to heal. The sores should be dressed on general principles of rest and antiseptic cleanliness. It will be necessary to use ointments or pastes to protect the surface and promote healing. Such preparations usually contain salicylic acid, resorcin, carbolic acid, or camphor or menthol. In all cases care must be taken not to use the antiseptic in too great strength. A useful dressing is—salicylic acid 2 parts, menthol 3 parts, soft white paraffin to 100 parts. Electric treatment of various kinds has been suggested for chilblain both in the early stages and later. In some cases the

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application of the high-frequency current relieves discomfort for a time, but of physical remedial measures those mentioned above, such as good hygienic conditions, good food, and appropriate exercises, are the most valuable.

JAMES GALLOWAY.

CHLOASMA (see PIGMENTATION).

CHLORAL POISONING (see POISONS AND POISONING).

CHLOROFORM POISONING (see POISONS AND POISONING).

CHLOROFORM POISONING, DELAYED (see VOMITING, POST-ANÆSTHETIC).

CHLORO-LEUKÆMIA (see LEUKÆMIA).

CHLOROMA (see LEUKÆMIA).

CHLOROSIS (see ANÆMIA).

CHOLÆMIA, FAMILY (see JAUNDICE).

CHOLECYSTITIS AND CHOLANGITIS.—These two allied conditions may be considered in the same article.

CHOLECYSTITIS

Inflammation of the gall-bladder may be either acute or chronic.

ACUTE CHOLECYSTITIS may be—

Catarrhal.
Suppurative.
Ulcerative.
Phlegmonous.
Gangrenous.

Etiology.—In all forms the direct cause is infection, which may arise from the bloodstream, or from the alimentary tract, especially the appendix, by the portal vein. In the former the commonest organism is *B. typhosus*, in the latter *B. coli*. Indirect causes are those which lead to biliary stasis, such as a sedentary life, obesity, tight lacing, pregnancy, or abdominal tumours.

Pathology.—In the catarrhal form the gall-bladder is distended, and its serous coat is dull and covered with fibrin. In the other forms of acute inflammation peritonitis is more marked. The mucous membrane is congested and covered with mucus, and the cystic duct may be occluded by the swollen mucosa. If the condition proceeds to suppuration, there is deeper congestion, and the contents are purulent. Single or multiple ulcers may appear.

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In *phlegmonous* cholecystitis the walls are swollen and oedematous and often infiltrated with pus. Pus is also present in the contents. Owing to the good blood supply, *gangrene* is rare. In such a case soft dark-green areas are visible in the wall. Perforation is common in both *phlegmonous* and *gangrenous* cholecystitis.

Symptomatology.—Slight cases of the *catarrhal* form, especially in enteric fever, often pass unrecognized; the more severe ones closely simulate dyspepsia or colic. Typical cases produce local tenderness and pain, which may be dull and continuous, or intermittent. The gall-bladder may be felt as a pear-shaped tumour, but the swelling may be masked by rigidity of the right rectus muscle and abdominal distension. Cutaneous hyperæsthesia of the eighth and ninth dorsal segments is almost constant. Respiration is shallow and jerky. Fever is absent or slight. The liver is not enlarged, and in the absence of cholangitis there is no jaundice. Local peritonitis may cause vomiting.

Suppurative cholecystitis is only distinguishable by the more marked local and general symptoms. The pulse is quicker, and fever and leucocytosis are present. After the formation of ulcers the gall-bladder usually becomes fixed by adhesions.

In *phlegmonous* and *gangrenous* cholecystitis the onset is sudden and the symptoms are those of peritonitis. Severe cases end fatally in two or three days, and in less acute ones a local abscess may form near the gall-bladder.

Diagnosis.—From *cholelithiasis* the diagnosis is often impossible, though a recent attack of enteric fever is in favour of simple cholecystitis. Typical gall-stone colic is more agonizing, whereas in *catarrhal* cholecystitis the local signs are more evident. The latter is often mistaken for *appendicitis*. The pain may be referred to the right iliac fossa, and if the liver lies low down the gall-bladder may form a swelling resembling that of appendicular abscess. With an inflamed appendix situated high up, a mistaken diagnosis of cholecystitis may be made. The two conditions not infrequently coexist. *Intermittent hydronephrosis* or *floating kidney* with Dietl's crises may imitate cholecystitis, but the great variation in urinary outflow in the one, and the elusive movable tumour in the other, should prevent confusion. *Pyelonephritis* is usually bilateral, but if right-sided may be mistaken for cholecystitis. Even without bladder symptoms there

will be albumin, pus, and organisms in the urine.

In acute suppurative, *phlegmonous*, and *gangrenous* cholecystitis the symptoms are those common to all acute abdominal conditions, and a more exact diagnosis is often impossible. It is most likely to be confused with *acute appendicitis*, but with a normally situated appendix the pain and tenderness are worst over the cæcum, whereas in cholecystitis the pain is worst near the right costal margin, and radiates upwards into the scapular region.

Prognosis.—In *catarrhal* cholecystitis this is good. The acute inflammation subsides quickly but often leads to chronic inflammation, cholelithiasis, chronic empyema of the gall-bladder, or local adhesions with attendant dyspeptic symptoms. The *suppurative* form rarely recovers spontaneously, and proceeds to local abscess formation or general peritonitis. The more acute cases of *phlegmonous* and *gangrenous* cholecystitis are rapidly fatal, the less acute are grave but do fairly well after early operation.

Treatment.—*Catarrhal* cholecystitis requires rest in bed and low diet. Pain is usually relieved by hot fomentations or leeches. If possible, morphia should be withheld, to avoid masking the symptoms. Mild cases require a laxative and hexamine (urotropine). Vomiting can be controlled, as a rule, by dilute hydrocyanic acid, tincture of iodine, or cocaine ($\frac{1}{10}$ gr.). For *suppurative*, *phlegmonous*, and *gangrenous* cholecystitis sips of liquid only are advisable. Morphia will be needed for pain and vomiting. Early operation is essential. For the suppurative form cholecystotomy with subsequent drainage is sufficient, but for *phlegmonous* and *gangrenous* cases excision of the gall-bladder is necessary.

CHRONIC CHOLECYSTITIS has the following forms:

- Membranous.
- Simple.
- Obliterative.

Etiology.—The membranous form is rare and is associated with membranous colitis. Simple chronic inflammation may follow acute cholecystitis, or the inflammation may be of low virulence from the beginning. Long-continued or recurrent attacks may cause partial or complete obliteration of the gall-bladder. Gall-stones are frequently but not necessarily present.

Pathology.—The gall-bladder in chronic cholecystitis is usually distended with mucus,

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which is often very thick or even semi-solid. Its walls are thickened. The gall-bladder in the obliterative form is contracted and distorted. Calculi are very common, and in these cases adhesions are often dense. The cystic duct may be closed by obliterative cholangitis. Chronic empyema of the gall-bladder is sometimes a sequel.

Symptomatology.—The symptoms of *membranous* cholecystitis are similar to those of gall-stone colic. In *simple* chronic cholecystitis there are recurrent attacks of pain without tenderness. *Obliterative* cholecystitis and empyema generally cause periodic pains and fever with gradual loss of health.

Diagnosis.—*Membranous* cholecystitis is recognizable only by the passage of bile-stained casts of the gall-bladder in the fæces. The symptoms of the other forms resemble those of *cholelithiasis*, but if the pain is not severe, and there is no jaundice, and no stone is found after an attack, the probability that they are due to cholecystitis uncomplicated by gall-stones is enhanced.

Prognosis.—This depends to some extent on the results of medical treatment, but, even if this fails, surgical treatment often restores the patient to good health.

Treatment.—Medical treatment consists in a light diet, with alkaline waters, and regular exercise. In the case of women loose corsets must be worn. The most useful drugs are morning salines and hexamine (antropine) or sodium salicylate. The best operative treatment is cholecystotomy followed by drainage if the cystic duct be found patent and bile be present in the gall-bladder; but if the duct be occluded, cholecystectomy is advisable.

CHOLANGITIS

Of inflammation of the bile-ducts, the following forms are recognizable :

Acute Cholangitis { Catarrhal.
 { Infective.
 { Suppurative.
Chronic Obliterative Cholangitis.

Etiology.—*Catarrhal* cholangitis may be due to an ascending infection of the duct secondary to alcoholic gastritis, duodenal ulcer, malignant disease, or pancreatitis. (*Catarrhal* jaundice is probably due to hepatitis, and is dealt with in the article on JAUNDICE.)

Infective cholangitis is nearly always associated with gall-stones (q.v.).

Suppurative cholangitis is either a sequel to cholelithiasis, carcinoma, ruptured hydatid

cyst, or some other obstruction; or is due to a primary infection such as enteric fever or influenza.

The etiology of *chronic obliterative* cholangitis is very similar to that of obliterative cholecystitis (see above), and they are often found together.

Pathology.—*Catarrhal* cholangitis is accompanied by swelling of the mucosa of the ampulla of Vater and bile-duct. The orifice of the duct may be plugged with mucus.

In *suppurative* cholangitis the liver is enlarged and smooth, while the bile-ducts are distended, especially near its surface. Multiple hepatic abscesses are generally found.

Symptomatology.—*Catarrhal* inflammation gives rise to symptoms of dyspepsia, to jaundice with pale stools and bilious urine, and often to some fever. It generally passes off within a month.

Suppurative cholangitis causes progressive enlargement of the liver, with tenderness and sometimes with pain. The spleen, too, is often enlarged. Jaundice may be absent, and depends more on the cause of the condition than on the suppuration. There is fever with rigors and sweats. In acute cases hepatitis with multiple abscesses develops, and in subacute cases a local abscess often forms. Suppurative pancreatitis or pyelephlebitis may be sequelæ, and pneumonia, pleurisy, and empyema are not uncommon complications.

Chronic obliterative cholangitis of the common or hepatic ducts causes progressive jaundice, which may become deep green in colour. In the early stages the liver is always enlarged, but later on it becomes smaller, and cirrhosis of the liver may be caused.

Diagnosis.—The history of previous illness and the recognition of the primary disease will generally serve to diagnose catarrhal cholangitis from *simple catarrhal jaundice*.

Suppurative cholangitis is distinguishable from *infective cholangitis* (intermittent hepatic fever) by the graver constitutional symptoms, the continuous fever, the absence of jaundice or its lighter and more constant character, and the absence of periodic attacks of pain. In *tropical abscess*, which is much commoner, there is usually a history of previous dysentery and none of gall-stone colic. Signs of local abscess are more likely to be present. *Carcinoma* may be a cause of suppurative cholangitis, so that it cannot be excluded even in the presence of obvious new growth. *Carcinoma* alone rarely

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causes so severe an illness. In *acute cirrhosis* there is a history of alcoholism and often of *hæmatemesis*, the spleen is larger and the constitutional symptoms are milder. *Pylephlebitis* differs in the greater frequency and earlier onset of jaundice.

Prognosis.—*Catarrhal* cholangitis generally clears up within two or three weeks of the onset. *Suppurative* cholangitis is fatal except in subacute cases with local abscess formation, and even these seldom recover. *Obliterative* cholangitis may cause little impairment of health for a long period, but toxæmia with or without cirrhosis causes death in the end.

Treatment.—*Catarrhal* cholangitis requires rest in bed, careful dieting, saline aperients, and alkalis or hexamine (urotropine). In *suppurative* cholangitis with local abscess formation the abscess must be drained and hexamine administered. In *obliterative* cholangitis attempts have been made to drain the hepatic duct into the duodenum, or, if the cystic duct be patent, to drain externally by the gall-bladder, but operations are generally unsuccessful.

F. A. COCKAYNE.

CHOLELITHIASIS (see GALL-STONES).

CHOLERA.—A specific gastro-enteritis, caused by the coma bacillus of Koch, endemic in India and the East, but occasionally spreading in epidemic form by the main lines of communication to Europe and other temperate countries, and characterized by watery vomiting and purging with resulting collapse, and often followed by uræmia.

Epidemiology.—In four of the six great European epidemics of the nineteenth century the disease travelled by the overland route from India through Afghanistan and Persia to Russia; in 1848 it was carried by sea, from Bombay to the Persian Gulf; and in 1865 and 1881 from the same Indian port to Mecca and Egypt. With improved communications the disease now travels more quickly than formerly, and in 1892 cholera spread from India to Russia by the overland route in five months, although the 1816-37 epidemic took as many years to cover the same ground. The disease is still a serious menace, especially to Eastern Europe, having caused high mortality in Russia in 1910; it was widespread in Italy in 1911, and during the Balkan wars in 1912-13. Fortunately it had nearly died out of Europe before the outbreak of the Great

War, during which only a few comparatively mild cases occurred in the Eastern areas. In 1919 a terrible outbreak in China was reported to have caused at least 300,000 deaths.

Etiology.—The comma bacillus was discovered by Koch in 1883 in Egypt. It is a slightly curved rod with a flagellum at one end, giving it motility. It is Gram-negative, liquefies gelatin along the inoculation streak and at the surface, and produces indol in peptone solution. Different strains show slight variations on culture, but a serum obtained from rabbits inoculated with a true cholera comma clumps other cholera strains in dilutions of 1 in 1,000 and upwards, but not harmless water and other saprophytic vibrios, thus serving to differentiate the specific organism. Certain cholera-like strains from comparatively mild cholera cases, when injected into rabbits, may produce serums which agglutinate them, but not true cholera vibrios. In severe cases of cholera the bacillus may be present in practically pure culture in the rice-water stools. When present in smaller numbers it can be separated by incubating in peptone water at 37° C. for a few hours and then plating on surface films of Dieudonné's alkaline blood medium.

There is abundant evidence that infection takes place through water and food containing the organism, flies sometimes conveying the infection from cholera evacuations. The disease is not otherwise infectious, for, as Ernest Hart well put it, "you can eat cholera and you can drink cholera, but you cannot catch it." Some of those who have recovered from an attack may harbour the organism in their intestines for from four or five to fifty or more days, and thus become carriers of the disease to distant places. In many of these carriers the gall-bladder is infected. On reaching New York a small number of immigrants from Italy in 1911 were found to be carriers.

In endemic areas there is a definite seasonal incidence. Thus, in its home in Lower Bengal the maximum occurs every year in the dry hot months of March to June, and the minimum in the rainy season from July to September. The case-mortality is highest in the earlier part of the maximum season and at the beginning of epidemics, and lowest at the end of an outbreak and in the rainy season in Bengal.

Pathology.—The changes found in the body after death are comparatively slight and very variable in degree. Pronounced congestion of

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the small intestines and sometimes also of the stomach, with much desquamation of the epithelium of the former, and occasionally patches of congestion or hæmorrhages in the cæcum, are the main changes, but if the patient dies in the late uræmic stage even these may be little evident. Sometimes the lymphoid follicles of the ileum and the Malpighian bodies in the spleen are enlarged. The kidneys are usually much congested and may show cloudy swelling and desquamation of the epithelium and, occasionally, intertubular hæmorrhages. Small patches of pneumonic consolidation may contain comma bacilli, as shown by Greig, who also found the bacillus in the gall-bladder in nearly one-third of fatal cases. The soluble exotoxins and the less powerful endotoxins, when absorbed into the system from the bowel, produce the symptoms of cholera.

Blood changes.—Of great practical importance are the blood and circulatory changes, as they can be studied during life, and successful treatment based on them. Owing to the great loss of fluid the blood becomes concentrated, the red corpuscles commonly numbering from seven to eight millions per c.mm. An actual leucocytosis is also present, with a low proportion of lymphocytes but an increase of the large mononuclears. The loss of fluid from the blood can best be estimated clinically by taking its specific gravity by means of a series of small labelled bottles containing glycerin and water of each two degrees of specific gravity from 1050, 1052, etc., up to 1070. These can readily be made up with the help of an accurate hygrometer, or even a urinometer. A small drop of the finger-blood is blown gently into the middle of one of the bottles by means of a capillary tube and detached. If it sinks at once it is heavier, whilst if it rises it is lighter. The bottle in which it floats for a second or two before finally sinking gives the required result. The solutions need renewing after prolonged use. Taking the normal figure at 1056-58, in cholera it will be found to have risen to from 1062 to 1068 or even higher. The immense value of this test will be seen when treatment is considered. The alkalinity of the blood is also greatly reduced in cholera, especially when uræmia is threatening. It is

commonly reduced in severe cases from $\frac{N}{80}$ and even to $\frac{N}{120}$; this also is a point of practical importance. Lastly, the salts of the blood, of which the chlorides form the great

bulk, may be lost to the body to even a greater extent than the fluid, and so set up a vicious circle by predisposing to renewed loss of any fluid supplied in the form of normal saline, thus explaining the comparative failure of this method. By injecting a solution containing a greater amount of salts than the normal blood, the fluid is much more likely to be retained and the excessive diarrhœa checked, as has been shown by the writer. The high salt content of the blood also helps to eliminate the cholera colloidal toxins from the system, as pointed out by Sir Benjamin Moore.

Symptomatology.—The onset is usually sudden, and often occurs in the early morning. In mild cases the first few stools may be composed of loose fecal matter—the so-called premonitory stage of diarrhœa. At the other extreme we have the very rare *cholera sicca*, in which the patient dies of collapse without any evacuation of the bowel, although after death the small intestine is distended with several pints of watery fluid, which has been effused so rapidly as to destroy life before the bowel can empty itself. In the great majority of cases, however, the stools are very copious from the first, and rapidly attain the typical appearance of water in which rice has been boiled, from which the term “rice-water stools” is derived. On standing, the white flocculi of desquamated epithelium separate and fall to the bottom, leaving an almost clear watery fluid above—a most characteristic appearance. Not rarely, in severe cases, the stools are pinkish from the presence of blood. The quantity of fluid passed is often astonishing, amounting to several quarts within two or three hours. Vomiting almost invariably accompanies the diarrhœa, and, after the contents of the stomach have been completely evacuated, almost clear water is expelled. Much fluid may be lost in this way, a quart being sometimes violently ejected at one time even when very little fluid has been taken by the mouth. The combination of colourless watery vomiting and diarrhœa is practically pathognomonic of cholera.

Collapse ensues in a large majority of the cases. The pulse becomes very feeble, and in about one-third of the cases is quite imperceptible at the wrist. This is accompanied by great restlessness (the patient being unable to lie still for more than a few minutes), cyanosis of the fingers and toes and, in severe cases, of the lips; and severe, frequently recurring, very painful muscular cramps beginning in

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the extremities, but extending to the abdominal muscles. The fingers become cold and shrivelled, and the eyes sunken. The axillary temperature, and even that of the mouth, may sink to 96° F. and even to below 95°, although the rectal temperature may be about normal. Death soon ensues if relief is not quickly afforded. If the rectal temperature also falls to 97° F. or below, the outlook is bad. In about one-third of sporadic cases, and towards the end of an epidemic, the serious collapse may not ensue, although the specific-gravity test will show considerable concentration of the blood, if pronounced anæmia had not previously been present. During collapse there is complete suppression of urine after the bladder has once been emptied with the first stools, and the longer this continues the greater the danger of uræmic complications during the stage of reaction.

Reaction.—In cases surviving collapse, after a variable time the stools become less frequent and copious, and may be somewhat milky in appearance. Bile next reappears, giving a yellow or green colour to the evacuations, which is a favourable sign. Warmth gradually returns to the extremities with the steady improvement of the pulse, and the cyanosis disappears. The stage of reaction, which has serious dangers of its own, is thus entered upon. The surface temperature rises above normal. This febrile condition is independent of any treatment, and may become excessive and end in hyperpyrexia, especially in hot damp weather. The reaction may end fatally even when the body temperature only rises to 103°–105° F., irrespective of the rise following intravenous injections of salines. These severe reactions appear to result from absorption of toxins from the bowel with the revival of the circulation.

Complications.—The most dreaded and frequent complication of cholera is continued *suppression of urine* after the circulation has been restored, death with uræmic symptoms inevitably following if the condition is not relieved within two or three days. In a patient otherwise doing well, complete or nearly complete failure to pass urine (retention in the bladder being excluded) is the most ominous sign in the reaction stage of cholera. The respirations increase in frequency and depth, the mental condition becomes obscured, and the pulse ultimately again fails. Estimations of the blood-pressure show that in many of these cases it is still considerably below normal, and

if it remains permanently below 80–90 mm. of Hg in an adult, it is due to partial vaso-motor paralysis, which prevents renewed secretion of urine. In cases of another class the blood-pressure is normal or in excess, yet uræmia ensues, which may sometimes be due to previous organic disease of the kidneys such as early fibrosis. Again, the secretion of urine may be deficient because the specific gravity of the blood is still in excess. Of still greater frequency and importance as a cause of post-choleraic uræmia is a condition of acidosis, which Sellards first detected in the Philippine Islands by noting the large quantities of alkalis which could be administered in such cases without the urine being rendered alkaline; he greatly reduced the death-rate from uræmia in this disease by adding alkaline carbonates to the saline solution injected intravenously.

Next to suppression of urine the most frequent and fatal complication of cholera is a *patchy pneumonia*, which may be difficult to detect during life. *Suppurative parotitis* occurs in about 1 per cent. of cases; the abscess should be opened early. In severe cases in asthenic subjects, *sloughing of the lower half of the cornea* and *bedsores or gangrene of a limb* may ensue. Sudden *heart failure* may supervene during convalescence if patients are allowed to sit up too soon. *Dysenteric symptoms* may succeed the diarrhoea, but usually yield readily to treatment.

Diagnosis.—The typical character of the main symptoms usually renders the diagnosis easy. In a small proportion of mild cases it may be in doubt for some time, and examination of the stools by culture may be necessary to establish it, including the agglutinating reactions of any comma bacilli isolated, for which expert laboratory assistance is essential. As these mild cases may spread the disease, it is important to be on the look-out for them whenever the occurrence of cholera is possible. Not rarely they first come under observation in the reaction stage with threatening uræmia. The diseases most likely to be confused with cholera are *ptomaine poisoning* and *intestinal malaria*. In these the typical rice-water stools are not seen, although the degree of collapse may equal that of severe cholera. It is fortunate that in ptomaine poisoning hypertonic saline injections are the proper immediate treatment, as only the progress of the case and bacteriological examinations can differentiate them with certainty. In intestinal malaria the specific gravity of the blood is usually below

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normal, leucocytosis is absent, and malarial parasites may readily be found in the blood. *Arsenical poisoning* may resemble cholera, and leucocytosis is present in both, but the gastric symptoms are more urgent in the former.

Prognosis.—Until a few years ago, cholera in Europeans, if severe and accompanied by pronounced collapse, was nearly always fatal. Fortunately, at the present time, if cases are seen in good time and correctly treated, the patient being not over 50 years of age and with sound kidneys, the mortality should not exceed 20 per cent. Taking all cases and races treated in the Medical College Hospital, Calcutta, and thus including late and complicated ones, the mortality under the system of treatment described below, among 638 cases, from 1915 to 1917 inclusive, was only 19·1 per cent, against one of 59·0 per cent. in the same institution during the eleven years before the writer began his investigations on the subject; a reduction of the death-rate to one-third of the former rate. The case-mortality rises with each decade in life after the second—from 15·4 per cent. in the second decade to 27·0 in the fourth and 50 per cent. in persons over 50 years of age. It is also high, 36·4 per cent., in children up to 5 years of age. Unfavourable signs are great cyanosis and restlessness, with complete failure of the pulse at the wrist and especially in the brachial artery; a specific gravity of the blood of over 1066; respirations over 40 per minute; rectal temperature as low as 97° F.; complete or almost complete suppression of urine for over forty-eight hours; a blood-pressure persistently below 90–100 mm. of Hg in an adult male, or slightly lower in a female; pneumonic consolidation; previous organic disease of the kidneys; very severe and prolonged fever in the reaction stage, tending to hyperpyrexia; and general asthenia with sloughing of the tissues.

Prophylaxis.—For the prevention of cholera, the boiling of all water and milk and the avoidance of uncooked food and vegetables are the most important safeguards. During the prevalence of the disease, disinfection of wells and other water supplies when possible with permanganate of potash is a valuable measure. The application of fresh chlorinated lime to stools and latrines is a valuable disinfectant measure with the great advantage of also keeping off flies. The cholera vibrios do not usually survive many days in faecally contaminated water, and they are very difficult to separate from water even during cholera prevalence.

Treatment.—No disease requires more vigilant and constant care in its treatment than cholera. There is certainly none which rewards the medical man with the saving of so large a proportion of valuable lives. We have seen that the great loss of fluid from the body is the essential factor to be combated, and that the specific gravity of the blood is the key to estimating that loss, and hence the amount of fluid to be replaced. Estimations of the loss of serum made with the hæmorrhite, and of the effect of rapid intravenous injections of saline solutions, have shown that a specific gravity of 1063 in a previously healthy subject means the loss of approximately three pints of fluid from the blood; one of 1064, four pints; and one of 1065, five pints. Even the last amount may occasionally be lost without serious collapse being present, although in such cases it is nearly certain to ensue before long. The first important rule is, therefore, that if the specific gravity of the blood is 1063 or over, the lost fluid must be replaced as soon as possible by **saline infusion**, even if collapse has not set in. The amount to be injected varies from three to six pints in an adult male, in accordance with the degree of concentration of the blood as shown by the specific gravity, and its effect on the pulse, which should be quite full and of good tension at the end of the little operation. It must be emphasized that cholera can only be treated efficiently with the aid of the specific-gravity test. In children of about 6, one pint can usually be injected with safety. Other useful indications of the need for immediate infusion are a weak or absent pulse at the wrist, a blood-pressure not exceeding 80–90 mm. in an Indian and 90–100 mm. in a European male, muscular cramps, restlessness, and cyanosis, and, during the reaction stage, continued deficient secretion of urine. Two solutions are used, one a hypertonic saline solution, of the composition given below, to counteract collapse, and the other an alkaline salt solution, to combat the acidosis which is so constant in severe cholera cases and predisposes to uræmia in the later stages of the disease. The hypertonic saline solution is composed as follows:—

Sodium chloride . . .	120 gr. (8 grm.).
Calcium chloride . . .	4 gr. (0·25 grm.).
Potassium chloride . .	6 gr. (0·4 grm.).
Water	1 pint (568 c.c.).

It is convenient to have the above in solid form of a size such that four to a pint form the

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hypertonic solution, and three an approximately isotonic one. The three chlorides are in the relative proportion of those in Ringer's fluid. The potassium salt is not essential, and for several years past I have omitted it on account of the possible depressant action on the heart of the large amounts that would be contained in the repeated copious intravenous injections often required in cholera.

The alkaline salt solution consists of—

Sodium chloride . . . 90 gr. (6 grm.).
Sodium bicarbonate . . 160 gr. (2 per cent.).

As the latter salt is decomposed by boiling to form the carbonate, 120-grain packets of the powder may be sterilized by dry heat or in an autoclave and added before use to a pint of the sterile salt solution. At each intravenous injection given in the collapse stage, one pint of the alkaline solution is used and the required total made up with the hypertonic salt solution.

Intravenous administration is by far the most effective method, and can alone be safely relied upon if collapse has set in, or if the blood has become very concentrated. The temperature of the fluid is most important. A rigor and fever practically always follow the injection, and fatal hyperpyrexia may result unless due precautions are taken. The coldness of the extremities during collapse appears, at first sight, to indicate the employment of a solution at above blood-heat, but the temperature in the rectum, which is usually normal or raised, is the correct guide to follow. If the rectal temperature is over 99° F. it may be dangerous to inject the fluid at above blood-heat, whilst if the rectal temperature is 100° F. or over, the solution should be given below blood-heat, e.g. at 90° F., or at the room temperature of the tropics, namely, about 85° F. The neglect of this precaution is the usual cause of dangerous hyperpyrexia after infusion in cholera. The rate of flow may be regulated by using a graduated glass vessel to hold the solution, and the present writer's stopcock transfusion cannula, the rate of flow being timed and the position of the stopcock altered accordingly. If the patient is collapsed or the blood much concentrated, the first three or four pints may be run in at the rate of 4 oz. a minute, and further amounts more slowly.

The immediate effect in a case of cholera is most striking. The agonizing cramps rapidly cease, the restlessness disappears, and the

pulse returns and becomes quite full. The cyanosis is removed, warmth returns to the extremities, and the patient is so relieved that sleep often comes on before the operation is completed. After the treatment with normal saline this great improvement usually lasted only a very few hours, as the diarrhoea increased and the replaced fluid was soon again lost. With the hypertonic solution the effect is commonly more lasting, although two or more injections are not infrequently required. The indications for subsequent injections are the same as for the original one, an increase of the specific gravity to several points above the normal and reduction in the blood-pressure being the most reliable. As many as six to ten injections, totalling 20–30 pints, may occasionally be given with ultimate success, but one or two usually suffice to tide the patient over the collapse stage.

The other methods of replacing lost fluid require only brief mention. Water and barley-water should be freely given by the mouth, and ice may be given to suck if the temperature is raised. Much of the fluid thus taken is lost through vomiting, but, if administered in small quantities frequently, a good deal is retained, and helps to assuage the awful thirst, whilst in cholera sickness does not usually cause much depression unless very copious. Rectal injections of the alkaline saline solution, half a pint every two hours, are most valuable, and in mild cases may suffice to stave off collapse and avoid the need for intravenous injections. The same measure should always be used to supplement intravenous injections, and be continued for two or three days until some two pints of urine are passed in twenty-four hours, but it may be given less frequently—every four hours—once urine is secreted in fair quantity.

Subcutaneous injections have also been largely used in the treatment of cholera, but they are far less effective than intravenous ones, and in the low state of vitality produced by the disease are only too often followed by abscess formation, even though carefully administered by experienced surgeons. They are also much more painful, and it is not safe to rely on them in a serious case. The hypertonic solution may be given subcutaneously, and is readily absorbed. Normal salines, 90 gr. of sodium chloride to a pint of water, given subcutaneously in quantities of one pint, are often of service if the urine is deficient in the later stages, but even then the alkaline intravenous

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injections are much to be preferred. Lastly, the intraperitoneal route has been used by the writer and others, but in Calcutta it has been abandoned on account of its being much less efficient and safe than the intravenous. It is sometimes of use in children with small veins, but the difficulty in these cases may be overcome by using a piece of glass tubing drawn out to a narrow point as a cannula, the internal saphenous vein at the ankle or in the thigh being selected if the veins at the bend of the elbow are too small. A puny infant of three months has been successfully injected at the ankle vein with a fine glass cannula.

Drugs per os.—In the collapse stage of cholera, owing to the failure of the circulation and damage to the mucous membranes, it is futile to give drugs by the mouth with the idea of their being absorbed into the system. Moreover, medicines given during collapse may accumulate in the alimentary canal and be absorbed, possibly in dangerous quantities, when reaction takes place. It is useless to try to stimulate the heart when the blood is too small in quantity and too thick to circulate. If the lost fluid and salts are replaced by sufficient hypertonic saline the circulation is at once restored, and if the diarrhoea is checked by the excess of salt retaining the fluid in the system, and if the toxins are eliminated, no further drug treatment is required. On the other hand, some danger arises through the absorption of toxins from the bowel in the reaction stage. The toxins are unstable forms of albumin, which are readily oxidized into harmless substances. For this purpose permanganate of potash, made up in 2-gr. pills with kaolin powder and a little vaselin, with or without a coating of keratin or of one part of salol in five of sandarach varnish, may be given. This addition produced a further reduction of the mortality in Calcutta hospitals, and has been favourably reported upon in district outbreaks in India, where often the full saline treatment cannot be carried out. Two of the 2-gr. pills are given every quarter of an hour for the first four hours in severe cases, and then every half-hour until the stools become green or yellow from the reappearance of bile. Upwards of 100 gr. of the drug are often given in a case without deleterious effects. In children a colloid permanganate is preferable, as it is tasteless and less irritating to the stomach. No other drug is required in the earlier stages. Opium and morphia should never be used, as they greatly predispose to

subsequent suppression of urine and fatal uræmia.

Reaction stage.—During the reaction induced by the intravenous saline the temperature of the patient must be closely watched, and if it rises to 104° F. or over, when it is usually accompanied by restlessness, cold sponging and cold-water rectal enemata should be resorted to at once. If unconsciousness accompanies the hyperpyrexia recovery never takes place, so it must be most carefully guarded against, especially during hot damp weather. On the other hand, a moderate rise to 100°–102° F. is a favourable sign, and even 104°–105° F. is not dangerous if promptly dealt with.

The other great danger in this stage is continued deficiency of urine threatening to lead to uræmia, for the prevention of which continued watchfulness is required. The specific gravity and blood-pressure should be taken morning and evening. If the former is not below normal, yet but little urine is being passed, the blood is still too deficient in fluid to enable the functions of the kidneys to be restored. One or two pints of the alkaline saline should therefore be injected, preferably intravenously, at a slow rate, from 1 to 2 oz. per minute. On the other hand, if the specific gravity is low, i.e. not over 1050, but the blood-pressure is under 90–100 mm. of Hg, in an adult male, vaso-motor paralysis is present and the condition is grave. Pituitrin every four hours hypodermically, caffeine-sodio-salicylate in 5-gr. doses every four hours, together with strophanthus or digitalin, are now indicated to raise the blood-pressure to the necessary height. Adrenalin alone is disappointing in this condition, its effect being too temporary, but it may be given alternately with pituitrin. The alkalinity of the blood in these cases will be found to be very low. Dry cupping morning and evening, and fomentations over the kidneys, are also useful in this stage.

The diet should usually be limited to water, barley-water, and coconut-water for two or three days in severe cases. Milk whey is then to be cautiously added, followed by farinaceous foods, and then milk. In weakly subjects 2 per cent. solution of glucose orally and by the rectum is often of value. Relapses are liable to result from too early feeding and necessitate another transfusion. The patient must be kept in the recumbent position for several days to avoid heart failure. Directly the strength begins to return, convalescence is surprisingly rapid in most cases, but a change and rest are necessary

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after such a severe illness before work is recommenced. It is not too much to say that the treatment described above has robbed cholera of nearly all its sufferings and most of its terrors.

LEONARD ROGERS.

CHOLERA INFANTUM (*see* DIARRHŒAL DISORDERS OF INFANTS).

CHOLERA NOSTRAS (*see* DIARRHŒAL DISORDERS OF INFANTS).

CHOLESTEATOMA (*see* OTITIS MEDIA).

CHONDRIITIS AND PERICHONDRIITIS (*see* LARYNX, CHONDRIITIS AND PERICHONDRIITIS OF).

CHONDRO-ARTHRITIS JUVENILIS DEFORMANS (*see* PSEUDO-CONALGIA).

CHONDRODYSTROPHY (*see* ACHONDROPLASIA).

CHORDEE.—Very painful erections, which come on during the course of an acute gonorrhœa. The penis is curved, the convexity being towards the abdomen. This is because the inflammation extends from the urethra to the surrounding tissue of the corpus spongiosum. The latter structure becomes rigid and incapable of the normal distension with blood during erection, but the corpora cavernosa lengthen to the usual extent. Large doses of bromide and energetic treatment of the gonorrhœa are the best ways of controlling the condition. Cold douches to the organ are of little use.

C. A. PANNETT.

CHOREA (*syn.* St. Vitus's Dance, Chorea Minor, Sydenham's Chorea).—A nervous disorder characterized by involuntary muscular movements and muscular weakness.

Etiology.—Chorea is now generally regarded as rheumatic in origin. This is borne out by clinical evidence or by the history in the majority of cases, although in a few instances no evidence is forthcoming, and the possible operation of other causes cannot be excluded. There is probably also a psychical factor, which may explain the facts that it occurs twice as commonly in girls as in boys, is sometimes an accompaniment of pregnancy, and is not infrequently brought into prominence by a fright. It is very doubtful if fright alone can cause it, and more often true that chorea renders the child more susceptible to fright and emotion. It may occur under 2 years of age, but is commonest between 6

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and 9. Post-paralytic chorea is a different condition (*see* Athetosis, under **ATHETOSIS AND TREMOR**).

Pathology.—The old view that chorea is due to showers of emboli into the cerebral vessels has been replaced by one that it is toxæmic or infective in origin, since the infective origin of acute rheumatism has gained credence. In most cases there is no source from which emboli could be derived, and the complete recovery which follows an attack makes the embolic theory improbable. Poynton and Gordon Holmes have demonstrated the presence of a cellular exudate in the perivascular lymphatic sheaths of the cortical vessels of the brain, and chromatolytic changes in the larger pyramidal cells and in the Betz cells. Such phenomena are compatible with a toxic cause, and moreover diplococci, resembling those described by the former as occurring in rheumatism, have also been found by them in the cerebral vessels and exudate as well as in the cerebro-spinal fluid, the blood, endocardium, pericardium, and joints. In one case they have been discovered in the brain tissue.

Symptomatology.—Commonly the first symptoms are mental, but they are generally overlooked. The child becomes petulant and headstrong, crying for no reason and impatient of control. Her writing becomes crabbed and crooked, and sums are ill-done. Careful examination at this stage may reveal an unbalanced pose and a little jerky movement of the face, the extended arms, or the legs. Such a condition may persist for weeks or even months before the disease becomes manifest.

Motor phenomena.—The involuntary (pseudopurposeful) movements vary greatly in degree from a slight twitching of the face or fingers to violent shock-like jerks of all the limbs, bringing with them a danger of bruises or other traumatic lesions. The twitching of the face makes it the mirror of all the emotions in turn. Speech may be slurred or jerky, or impossible, whilst involuntary clucking noises are often emitted. The twitching of the fingers leads to the dropping of objects. Contractions of the trunk muscles may cause a "squirming" of the whole body. The movements are more marked on one side in a large proportion of cases, but true hemichorea with absence of movements altogether on one side is very rare. Intercostal and diaphragmatic breathing may be dissociated, and sighs are often heard. Some degree of muscular weakness is almost always present, so that the child crouches when

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sitting and the grasp is weak. In certain cases, known as "paralytic chorea" or "chorea mollis," the weakness may be extreme, the muscles are very atonic and soft, and almost all power is lost. In these, aphasia, loss of knee-jerk, and incontinence are sometimes met with. If the arms are held above the head, wrist-drop may be noticed, whilst in the more common cases hyperextension of the wrists occurs. The external ocular muscles are seldom affected, but hippus and dilatation, inequality, or eccentricity of the pupils are often met with.

Sensory symptoms are not usually detectable, though areas of hyperalgesia and of anæsthesia are sometimes found.

Reflexes.—The knee-jerks may be increased. Sometimes, when the patellar tendon is struck, two or more contractions are noticed, or a spasm may be induced, the leg remaining extended for a considerable period and the so-called "hung-up" jerk being produced. The knee-jerks, as well as the abdominal reflexes, may be lost in paralytic chorea and in severely toxic cases. The plantar response is not infrequently extensor.

Mental symptoms.—The child is always unbalanced and prone to immoderate tears or laughter. In severe cases the cries are quite characteristic, resembling the howling of an animal. Hallucinations and maniacal symptoms sometimes occur.

Cardiac signs.—Increased rate, arrhythmia, weakness of the first sound, and a systolic murmur inside the apex are often present; they constitute part of the clinical picture of chorea and serve to indicate its rheumatic origin. Less debatable are such associated rheumatic lesions as valvular disease and myocarditis with dilatation, which are not uncommon accompaniments, and pericarditis, which is rare. The gradual production of mitral stenosis in slight but chronic chorea is of importance.

Toxæmia.—In severe cases there may be cyanosis, sordes of the lips and tongue, coated tongue, torpor, prostration, dry, cracked hands, and a rise of temperature—toxic symptoms, indicating active rheumatism.

Diagnosis should be easy in all but exceptional circumstances, yet chorea is continually being confused with *tic* or *habit spasm*. In this, the same movements are repeated and are usually confined to certain groups of muscles, producing a blinking of the eyes, shrugging of the shoulders, etc. The move-

ments of chorea are general, and always varying. The irregular, bilateral, jerky and, sometimes, rhythmical movements of the limbs which occasionally accompany the onset of *vertical meningitis* have been mistaken for chorea, but within a few hours the diagnosis is no longer in doubt. Confusion may be occasioned with exceptional cases of *cerebral diplegia*, when the latter includes among its symptoms choreiform movements affecting all the limbs, and jerking of the body. In diplegia the protracted history, the association of fits and mental deficiency, and the coexistence of spasticity are distinguishing features.

Prognosis.—In cases of moderate severity the symptoms subside after about six weeks, but in paralytic, maniacal, and toxic cases they may continue for several months. Mild cases also are protracted. Sometimes the condition improves until only slight evidences remain, but these are very persistent. With the mild cases they constitute what is known as "latent chorea," which may continue for years and is liable at any time to develop into a more severe attack. A fatal issue is quite exceptional, and usually results from exhaustion or associated heart disease.

Treatment.—Bed is essential except in the latent cases; if it is accompanied by very active movements the bed should be padded to safeguard the child from injury, and in the meantime she can rest on a ample mattress on the floor. Excitement must be avoided by screening the bed if necessary. The diet should be plentiful, but if there is difficulty in mastication it should contain no hard particles. In severe cases food in jelly form, such as junket, custard, meat or fruit jellies, is indicated, and is swallowed more easily than fluids. Rarely, in the severest cases, feeding by tube may be necessary.

Drug treatment is of small importance in chorea, and no drug can be considered specific. Sodium salicylate 5–10 gr. t.d.s., or aspirin in somewhat smaller doses, treats the underlying rheumatism, and is essential if there is any rise of temperature. Arsenic has been much in vogue, and in the form of liquor arsenicalis has been recommended in large doses, but its effects are indeterminate, and the danger of arsenical neuritis is a very real one. Sedatives such as bromides, chloral (5 gr.), and trional (3–5 gr.) are especially useful if sleep is interfered with; when exhaustion threatens they are

CHOREA, HUNTINGTON'S

very valuable in larger doses, especially if accompanied by a warm pack. Chloretone is recommended by Wynter; it should be used with caution, a strict watch being kept on the degree of drowsiness and for the appearance of rashes—an indication that it should be abandoned. A combination of strychnine and ergot was advocated by the late Eustace Smith, but its use has proved disappointing. A. L. Goodman has recommended the intrathecal injection by lumbar puncture of an autoserum; 50 c.c. of blood is removed from the patient's median-basilic vein, and up to 20 c.c. of the separated serum injected. This may be repeated at intervals of a few days. When the active stage of the disease is over, tonics containing iron are beneficial; and if wasting has occurred, one of the preparations of cod-liver oil and malt is indicated.

Recovery may be hastened by massage and exercises, particularly those devoted to the correction of inco-ordination. At first such crude and easily performed exercises as apposing the fingers of one hand to the corresponding fingers of the other, or applying the heel of one foot to the ankle, shin, or knee of the other leg, may be employed. Afterwards a cribbage board, draughts, dominoes, or cards provide opportunities for systematic education in precision of movement. When the patient is up and about, chalked lines or dots and patterns of the carpet afford means of improving her gait. These simple devices materially hasten recovery, if their employment is deferred until the active stage of the disorder is over.

FREDERICK LANGMEAD.

CHOREA, HUNTINGTON'S (*syn.* Hereditary Chorea).—Although the movements in this disease resemble those of true chorea to some extent, there are certain definite distinctions between the two conditions. Huntington's chorea is hereditary and familial, begins between the ages of 30 and 40, the movements are slower and less jerky, there is more inco-ordination, and mental deterioration is present. Gradually the disease progresses until the patient becomes bed-ridden. Slight spasticity may be detected in advanced cases. The mental condition, at first emotional with periods of excitement and suicidal tendency, later becomes one of complete dementia. Chronic diffuse encephalitis and meningitis, and also primary degeneration of the cortical cells, have been found after death.

FREDERICK LANGMEAD.

CHORION-EPITHELIOMA

CHOREA, HYSTERICAL (*see* TICS AND HABIT SPASMS.)

CHOREA, POST-PARALYTIC.—One of the rarer late results of hemiplegia, manifesting itself in irregular involuntary movements in the limbs which have been affected. Occasionally they resemble the involuntary movements of chorea, but usually they are more correctly described as athetosis or tremor. (*See* ATHETOSIS AND TREMOR.)

CHORION-EPITHELIOMA (*syn.* Decidua Malignum; Syncytioma).—A malignant growth arising usually in the uterus, sometimes in the Fallopian tube or ovary, but always following upon a pregnancy. It is now known to be derived entirely from foetal structures, and to be a product of the coverings of the chorionic villi, the trophoblast.

Etiology.—One of the most striking facts is that in about half the recorded cases it has followed upon the expulsion of a hydatidiform mole. It has long been known that hydatidiform mole in some circumstances may penetrate deeply into the substance of the uterine muscle, and it seems certain that chorion-epithelioma arises from fragments of degenerate chorionic villi thus left behind in the muscle after the expulsion of a mole. The other cases of chorion-epithelioma have followed abortion or labour, and there is no authentic case on record in which an antecedent pregnancy could be excluded.

Pathology.—The degree of malignancy exhibited by a chorion-epithelioma is variable, but as a general rule it must be regarded as very high. The malignancy is evidenced by infiltration and perforation of the uterine muscle, and the formation of secondary growths, identical in character, in all parts of the body. There is a special tendency to form secondary growths in the vagina, probably by means of direct transplantation of minute fragments of growth from the uterus upon small injuries or abrasions of the vaginal wall. After the vagina, secondary growths form most commonly in the lungs, but no organ in the body is exempt.

The growth is recognized in the uterus by its forming a hæmorrhagic mass like a blood-clot, involving the uterine muscle. It practically never forms any large mass of white or yellowish growth like other malignant tumours; indeed, the actual amount of new growth formation is always small, and the bulk of the tumour seen by the naked eye is made up of

CHORION-EPITHELIOMA

bloodclot. The secondary growths have the same character. This hæmorrhagic structure is due to the special tendency which trophoblastic tissues have to attack and open blood-vessels, and consequently hæmorrhage into the growth and externally is a constant accompaniment.

Microscopically, the tumour is composed of elements derived from the syncytium and the Langhans cells, which together make up the trophoblast. At no time is there any stroma or connective tissue in it.

Symptomatology.—The chief symptom is a continuation of hæmorrhage after the expulsion of a mole, an abortion, or a full-term foetus. Sometimes there is a short interval during which the lochial discharge stops, but it then comes on again and becomes profuse. With bleeding there is often an offensive discharge because decomposition occurs in the masses of bloodclot which occupy the uterine cavity. A certain amount of pain is caused by uterine contractions which occur from time to time and attempt to expel clots from the uterus; otherwise pain is not a marked feature. In some quite unusual cases there is a long interval of time between the last known pregnancy and the appearance of symptoms of the new growth. In these cases probably a fresh very early pregnancy unrecognized by the patient is really the starting-point.

Diagnosis.—Chorion-epithelioma is usually mistaken for *subinvolution of the uterus* with retained products of conception. The diagnosis cannot be made out with certainty until an exploratory curettage of the uterine cavity has been carried out, and the material obtained has been submitted to microscopical investigation. Some hint might, however, be obtained if the uterus could be made out to be enlarging from week to week, instead of remaining stationary as it would with simple retention of conception products. The presence of a secondary growth in the vagina has sometimes been the first feature to call attention to the true nature of the case.

Prognosis.—Until something more is known of the distinctive characters of those chorion-epitheliomata which are highly malignant, as compared with those which are relatively benign, all such growths must be regarded as malignant in the highest degree, and treated as such. The duration of life after a chorion-epithelioma has been diagnosed is most variable. In the worst cases death occurs within about four months, secondary growths appear-

CLEFT PALATE

ing very rapidly and growing actively in spite of removal of the primary tumour. In comparatively benign cases simple curettage of the uterus has been said to have resulted in a cure, in one case even after the removal of a secondary growth in the vaginal wall.

Treatment.—Since the growth always starts in the uterus, and must be regarded as malignant, the only treatment likely to be of use is total hysterectomy. Removal of secondary growths is rarely possible, except those in the vaginal wall.

THOS. G. STEVENS.

CHOROID, AFFECTIONS OF (*see* UVEAL TRACT, AFFECTIONS OF).

CHROMIDROSIS (*see* SWEAT-GLANDS, AFFECTIONS OF).

CHYLOUS ASCITES (*see* ASCITES).

CHYLURIA (*see* FILARIASIS; URINE, EXAMINATION OF).

CIRCUMCISION (*see* PHIMOSIS).

CIRRHOSIS OF LIVER (*see* LIVER, CIRRHOSIS OF).

CIRRHOSIS OF LUNG (*see* LUNG, FIBROSIS OF).

CIRSOID ANEURYSM (*see* ANEURYSM, CIRSOID).

CLAUDICATION, INTERMITTENT (*see* ARTERIAL DEGENERATION).

CLAUSTROPHOBIA (*see* PSYCHASTHENIA).

CLEFT PALATE.—The malformation may be complete or incomplete. If the former, the alveolus and the whole length of the palate is cleft, and as a rule hare-lip is present. When the hare-lip is unilateral the cleft in the alveolus is between the maxilla and premaxilla, and is in the midline in the hard palate. Anteriorly, the septum nasi is adherent to the horizontal process of the maxilla. The cleft follows the midline in the soft palate. When the hare-lip is double and complete on each side, the cleft in the palate is mesial, except in front, where it is continued on each side of the premaxilla. The lower margin of the septum nasi is between and unattached to the margins of the cleft.

Incomplete clefts may affect (a) the uvula only, (b) the uvula and soft palate, or (c) the uvula, soft palate, and a greater or less extent of the hard palate. The cleft is always in the middle line. A careful examination of the

CLEFT PALATE

alveolus will often reveal a slight vertical ridge or depression, or an irregularity in the alignment of the incisor teeth on one side.

Congenital clefts of intermediate portions of the palate are very rare, and when these defects are present they should always be investigated carefully from the point of view of causation—injury and syphilitic affections being much more often the cause of such holes.

The **diagnosis** of the congenital nature of the cleft is generally easy. Difficulty sometimes arises when the patient is seen for the first time later in life, and the presence of scar tissue has interfered with the movements of the soft palate. Such scar tissue may have resulted from disease or injury, or from an operation to close the defect. There may be no history to aid in making the diagnosis. A congenital cleft of the soft palate is always in the middle line, the two halves of the uvula are equal in size and the palate is freely movable. The presence of linear longitudinal scars on each side just inside the line of the alveolus would suggest some previous operative measure to close the defect. As a result of syphilitic disease the uvula is wholly or partially destroyed; and the same applies to the soft palate. In these cases, also, adhesions to the pharyngeal walls are generally present. A round or oval hole with thin edges at the junction of the hard and soft palate, with limited mobility of the latter, is very suggestive of syphilis.

Treatment.—The worst effect of cleft palate is the interference with speech; it is the soft palate which is so important in this connexion. Clefts in the hard palate can be remedied by obturators, but no instrument can replace the delicate movements of the soft palate which are essential to proper articulation. Hence the essential point in treatment is to give the patient a movable soft palate—a palate which is as nearly a normal one as possible. In many cases, besides being cleft, the soft palate is too short to shut off completely the postnasal space; therefore, although the palate as the result of an operation may look normal and be freely movable, some defect in speaking may remain.

An infant with cleft palate is often difficult to feed, but provided that care is taken it need not suffer from malnutrition. A simple method is to allow the milk to pass slowly into the hollow of the cheek from a spoon, or the child may be fed from an ordinary boat-shaped bottle to which a rather wide teat,

with a hole on its under aspect, is attached. By gently tilting the bottle, the milk is allowed to flow slowly and intermittently into the mouth.

It need hardly be pointed out that the child's mouth should be in a healthy condition before the operation is done, but the indiscreet removal of a small amount of adenoid tissue is to be deprecated. Moreover, the general health and nutrition of the child should be as good as possible—the better these are, the greater will be the thickness of the mucoperiosteum of the palate. Decayed teeth should be removed.

Much discussion has taken place as to the best time and method of operating. There is no doubt that the defect can be closed immediately after birth by the flap method advocated by Sir Arbuthnot Lane. Brophy's operation aims at approximating the bones by means of wires passed through the upper jaws, tightening the wires, and so bringing the edges of the cleft together. This operation is performed at three to six weeks after birth. The writer is in favour of operating by Langenbeck's method, and the best results are obtained when the child is some months old. The exact date when the operation should be done is determined by the general health of the child, the extent of the cleft, and more particularly its height and width. It should always be done before the child has learnt to talk. There is no doubt that the cleft diminishes in width as the child grows, and the amount of tissue available for the flaps is increased.

Postoperative treatment.—Every endeavour should be made to prevent the child from crying. Hunger and thirst are potent causes of crying: drinks of water or feeds of milk may be given—a couple of ounces may be enough—the quantity varying with the age. Liquid foods should be given at frequent intervals, every two or three hours, and a drink of water to cleanse the mouth after each feed. At the end of a week, if the child is old enough, bread and milk (without crusts), custard pudding, and the like may be given. Immediately after the operation, sedatives, e.g. potassium bromide, may be necessary. Irrigation of the mouth is not usually required, but boric-acid lotion or bicarbonate of soda solution (10 gr. to 1 oz.) may be employed when necessary. If there is fætor, weak carbolic lotion (1 in 100) may be used.

The stitches may be removed at the end of ten or fourteen days; it is generally advisable

CLEIDO-CRANIAL DYSOSTOSIS

to remove them by stages, and an anæsthetic may be necessary. In the case of older children, talking should be prohibited until after the stitches have been removed.

Postoperative complications.—Vomiting is not infrequent during the first few hours after the operation; if persistent, bicarbonate of soda should be given. Protracted oozing of blood sometimes takes place. If it becomes serious it is best treated by pressure applied by means of tampons of gauze, or by giving an anæsthetic and packing the lateral incisions with gauze. Sloughing of part of the flaps and failure of union may also occur; both are usually partial.

Late treatment.—As soon as the child begins to talk, every care must be taken to train him to speak properly. He should be taught to speak slowly and with precision, and to pronounce each word and consonant properly. The consonants P, B, T, D, K, G, and combinations of consonants are most difficult. Months and years of careful supervision by parents and teachers will be required to obtain the best functional results of a successful operation.

Dental treatment is frequently required to correct any deficiency, decay, or want of proper alignment of the teeth. Breathing exercises are also useful.

Nowadays obturators are rarely, if ever, used in the treatment. Very few children can tolerate them, and cases in which it is impossible to close the soft palate are exceptional. They are only useful to cover up holes in the hard palate which it is impossible to close otherwise; they should never fit in the hole, but have the form and shape of a denture.

T. P. LEGG.

CLEIDO-CRANIAL DYSOSTOSIS.—

This is a rare congenital deficiency characterized by an absence or imperfect development of the clavicles and defective ossification of the bones of the cranium. It is congenital, hereditary, and more than one member of a family may be affected. The patients are usually undersized, but are not deficient mentally.

The clavicles may be absent, but, more commonly, their sternal ends are present, the remainder being represented by a ligamentous structure. Sometimes the outer thirds, sometimes the middle thirds, are the parts which are lacking. Want of development of the muscles is found, and varies according to the form of abnormality of the clavicles. Owing

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to the clavicular defect, the shoulders may be made to meet in front of the sternum.

In the skull the chief variations from the normal are these: The anterior and posterior fontanelles are widely open, or together may form one large unossified area. Closure may not take place for twenty years, and sometimes is never attained. The frontal, parietal, and occipital bones on each side are bossed. The horizontal measurements are increased, but the vertical diminished. Development of the superior maxillary bones is imperfect, the palate being high and arched, and often cleft. Slight exophthalmos may result from under-development of the floor of the orbit. The maxillary sinuses are small, while the frontal sinuses are prominent. Abnormalities of the teeth are common; they appear late, are ill-formed, and soon become carious; some may be absent. Curvatures of the back and other deformities may be associated. Many cases show only part of the changes seen when the condition is complete.

The osseous deficiency causes no disability, and treatment is unnecessary.

FREDERICK LANGMEAD.

CLIMACTERIC, SYMPTOMS OF (see MENOPAUSE).

CLIMATE IN THERAPEUTICS.—In the following brief outline of climatological therapeutics an attempt has been made to place before the reader those facts which seem both well authenticated and practically useful.

One point, common to all branches of the subject, must be emphasized at the outset, viz: that *whoever seriously needs treatment by climate needs also medical supervision*. If this principle be not borne in mind, advice otherwise excellent will, sooner or later, bring discredit on the adviser.

It should also be remembered that arrangements for the journey, provision against fatigue and cold, suggestions for suitable clothing, and the choice of hotel or rooms to be occupied on arrival, are all things to be thought of.

Anæmia of a simple kind may sometimes be cured by climate. A bracing place is best, usually inland and at a moderate height; but a tiring journey should be avoided. In slight cases the Alps are useful, as the effects of altitude on the blood might lead us to expect; in severer cases, however, great heights may be badly borne. The seaside suits some

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patients, and, for good sailors, in good weather, a sea voyage may be suggested.

Asthma (the true nervous type) is notoriously capricious. Young people generally gain most climatically from long residence in the Alps, especially at Davos and Arosa. At home the following localities are of service, viz. Hindhead, Haslemere, Malvern, the Farnborough and Bagshot districts, Broadstairs, Worthing, Ventnor, Bournemouth, Lyme Regis, Budleigh Salterton, the higher part of Exmouth, Torquay, Paignton, Newquay. Abroad, for winter, we may advise Arcachon, Pau, Hyères, Mentone, Algiers, Algeiras, Santa Cruz in Teneriffe, or Grand Canary. In summer a sea voyage is sometimes successful. Some asthmatics suffer least in large towns like London.

Bronchitis (chronic).—The climatic conditions to guard against are damp, cold, and wind. Winter, therefore, should be spent in a warm, sunny climate. If there is much sputum, dry air generally agrees best, and Egypt (Mena House, Helouan, Assouan) or the Riviera may be recommended, the patient not returning to England until May. If, on the other hand, the bronchitis is dry and irritable, a moist, warm, equable atmosphere is indicated, and for such cases there is probably no better climate than that of the south coasts of Devon and Cornwall in sheltered places; Sidmouth, Torquay, Falmouth, and Penzance are all good. So are, in Ireland, Rostrevor, Queenstown, and Glengariff. If the subject of dry catarrh, however, wishes to go abroad, he may choose Madeira, Ajaccio, Pau, Algiers, or Arcachon. Of home climates which are less moist, those of Bournemouth, Ventnor, and Hastings are useful. In summer a change may be made inland to Malvern. For young patients high mountain climates are often of great value.

Convalescence.—After acute disease there is usually no more useful means of hastening recovery than change of air. The place will vary with the nature of the illness, the idiosyncrasies of the patient, and the season. Thus after measles and whooping-cough, which predispose to tuberculosis, bracing but sheltered places are indicated; after rheumatic fever, warm, dry, sunny places; gouty patients as a rule do better inland than by the sea. In winter most of the English bracing resorts are too bleak, and the warmer seaside towns on the south coast may be advised, or, abroad, Biarritz or the Riviera. Summer is the time when *British bracing climates* are most in re-

quest, and it may not be superfluous to indicate where the chief of these are to be found. Probably the most bracing district in the British Isles is the north-east of Scotland, with the coast towns of Dornoch, Nairn, and Peterhead, and the inland mountain valleys of the Spey and Dee, where Grantown, Kingussie, Nethy Bridge, Aviemore, and Braemar are situated. Next comes the Scottish east coast farther south, with St. Andrews and North Berwick; next, the whole English east coast, with Redcar, Saltburn, Whitby, Scarborough, Filey, Bridlington, Hunstanton, Sheringham, Cromer, Mundesley-on-Sea, Yarmouth, Lowestoft, Southwold, Aldeburgh, Felixstowe, Walton-on-the-Naze, Clacton-on-Sea, Southend, Westgate, Margate, Broadstairs, Ramsgate, and Deal. Probably the north-east Irish coast about Portrush should be classed with these. Still bracing, but less so, are the towns in the eastern half of the south English coast; Folkestone, Hastings, Eastbourne, Brighton, Worthing, Littlehampton, Bognor, and Southsea; and the north Cornish coast, with Newquay, Bosccastle, and Bude. Weston-super-Mare is also valuable.

Inland also the English and Welsh hill country contains many bracing districts, as round Ilkley, Harrogate, Buxton, Llandrindod Wells, Malvern, Crowborough, Hindhead, Heytor, Chagford, Okehampton, and Yelverton.

In colder weather more westerly south coast resorts are available. The more bracing are Swanage, Weymouth, Lyme Regis, Seaton.

Abroad, Switzerland provides some of the most invigorating places in Europe—especially at heights over 5,000 ft. Such are Mürren (5,348 ft.), Engstlenalp (6,033 ft.), the Eggischhorn Hotel (7,362 ft.), Belalp (7,155 ft.), and Arolla (6,572 ft.) in Western Switzerland, and the Engadine (5,600–6,090 ft.) in Eastern Switzerland.

Dyspepsia (chronic) is often benefited by change to a bracing climate with outdoor interests and exercise. Dry inland places amongst mountains are perhaps best.

Gout (chronic).—When ordering change of air to gouty persons it is well to remember that sea-air often does not suit them, and that dry inland resorts of moderate elevation generally agree well.

Graves's disease (chronic) is sometimes improved or cured by prolonged residence, *with rest*, in high altitudes. These patients do not stand heat well, and a bracing place is desirable.

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Heart disease must be very cautiously dealt with. Premature change of air may be gravely injurious in recent rheumatic cases. But when the lesion is stationary, change of air may much improve the cardiac tone. A dry atmosphere with little wind, a dry soil with level walks, plenty of sun, and a house so situated that the road home is not uphill, are the requisite conditions. In bracing air the heart shares in the general invigoration, but heights of over 3,000 ft. are seldom desirable and, for elderly, weakly, or ill-compensated cases, are inadmissible. The mild equable climate of south-east Devon affords, in sheltered places, particularly good winter quarters, where bronchitis, renal complications, and rheumatic fever are all uncommon.

Insomnia.—The south-western peninsula of England is often useful—South Devon and South Cornwall perhaps particularly so. Mere change of air and scene, provided the climate is not an exciting one, go far towards restoring sleep. It will be well to inquire beforehand about the beds, which in some hotels are very bad.

Laryngitis (chronic), if dry and irritable, will probably derive most benefit from the warm, moist, equable atmosphere of South Devon and South Cornwall, in sheltered places; or abroad at Ajaccio, Algiers, Madeira, Mentone or San Remo.

Malaria (chronic).—High elevations with a dry, sunny atmosphere, especially near glaciers, are found beneficial.

Nephritis (chronic).—If the patient is fit to travel, a warm, dry climate, with little wind, and with access to a good milk supply, may be advised for winter. Egypt has perhaps the best climate, the Riviera the next best. But health resorts on the south coast of England, such as Bournemouth, Sidmouth, Exmouth, and Torquay, are far from negligible. These English resorts also do well for all-the-year-round residence. High altitudes in the Alps are injurious.

Neuralgia improves in a dry bracing place, fairly sheltered. Some patients get worse at the seaside.

Neurasthenia.—A well-built patient with good circulation will probably find a bracing seaside or mountain resort best. For such the high Swiss Western Alps or the Engadine may be recommended. A weakly person with feeble circulation may benefit more from a quiet inland country place. In some cases a sea voyage works wonders, but for depressed

and sleepless persons with any suspicion of melancholic tendency, a voyage cannot be too strongly condemned, on account of the temptation to suicide. The therapeutic value of intellectual enjoyment should not be lost sight of, and places of historic and artistic interest may do much to aid recovery, *if not allowed to induce fatigue*. Among such places are, in winter, southern Spain, Rome, Naples, and southern Italy, Sicily, and Egypt; in early summer and autumn, Spain generally, Venice and northern Italy, Florence, Vienna, Athens; in June and early July, Munich, Dresden, Switzerland, the Tyrol, the Rhine country, Belgium, Holland, and "the northern capitals."

Paroxysmal hæmoglobinuria should, when possible, be treated by transfer to a warm climate for the winter.

Phthisis.—In choosing a locality for a phthisical patient certain climatic conditions should be carefully avoided. These are—

1. The tropics, except at great altitudes.
2. The immediate sea front (by which, however, is not meant the coast in general; removal a quarter of a mile inland seems to take away whatever bad effect the actual seashore produces).
3. A damp soil.
4. Impure or dusty atmosphere.
5. Deficient air supply.
6. Deficient sunlight.
7. Fog.
8. Exposure to strong prevalent winds, especially rainy winds.

When the great value of open-air methods of treatment became generally understood, it was taught for a time that "sanatoria" had superseded all considerations of climate. Now it is becoming evident that this is not the case. Since sanatorium treatment by no means cures all cases, no reasonable adjuvant can be safely dispensed with. A survey of the results of sanatoria differently placed also suggests the idea that varying success may be due in part to varying location. The earlier sanatoria, such as Brehmer's at Görbersdorf, were built in carefully chosen situations; Görbersdorf is in a very sheltered valley among the eastern foothills of the Riesengebirge, at a height of 1,700 ft. and in a district formerly stated to be free from consumption. In the light, therefore, of these considerations it is necessary to give renewed attention to influences which earlier observations tended to prove important.

Chief amongst these is *altitude*. Some

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twenty years ago nothing was better attested than the great value, in suitable cases, of the treatment of phthisis at certain high-altitude stations, notably at Davos (ca. 5,100 ft.). More recently it has been alleged that altitude makes no difference in the result. Obviously this is a matter which needs careful re-examination. Professor Sahli, of Berne, has been so good as to send me, on this point, the result of his great experience. He writes: "If I had to choose, I should prefer the altitude climate without sanatoria, to sanatoria without climate. According to my personal experience," he adds, "there is a very great difference between the results of sanatorium treatment in high and in low countries. I have seen many cases improve only in Davos or Arosa after having long been treated before without success in German low-placed sanatoria."

A close scrutiny of the results of sanatorium treatment at home and abroad during the past decade and of the earlier results and conclusions from various climates leads me (whilst fully admitting, indeed urging, the necessity for a thorough revision of our knowledge in these directions) to sum up as follows the advice which may be most safely given in phthisis.

CLASSIFICATION OF PHTHISIS CASES.—For practical purposes we may divide our patients thus:

1. Those who should be kept at or near home.
2. Those who may be sent away.
3. Those who must decide the question for themselves.

In considering the last two classes we must further distinguish between the rich and the poor.

1. Phthisis patients who should be kept at or near home.—No one would be likely to send away patients with acute miliary tuberculosis, acute disseminated tuberculous pneumonia, or acute pneumonic phthisis in its acute initial stage. One would not advise the subjects of advanced phthisis with albuminuria, phthisis with ulcerative diarrhoea, advanced laryngeal phthisis or diabetic phthisis to leave the comforts which home, or a hospital within reach of their friends, can alone provide for them. Rapidly advancing phthisis and phthisis with persistent high temperature, although they may derive advantage from a well-placed and not distant sanatorium, are unsuitable for long journeys. Setting these cases, therefore, aside, we turn to those for which the question

of change of climate may be reasonably entertained.

2. Phthisis patients who may be sent away.

—If a patient be already living in a suitable climate and showing signs of improvement, his doctor will hesitate before advising him to change it. In any case, also, where climatic change is contemplated, it is probably advisable, if considerations of season permit of delay, to give a preliminary course of sanatorium treatment, partly to gain as much knowledge as possible of the tendency of the case, partly to educate the patient into the avoidance of imprudences.

Consider, first, *foreign climates as we can order them for the wealthy*. For practical purposes we may enumerate the following varieties of cases:

- (1) Early cases, young, vigorous, uncomplicated, with little or no fever.
- (2) Early cases with recurrent hæmorrhages, i.e. not from cavities.
- (3) Early cases which have originated in pleurisy or pneumonia.
- (4) Early cases with little or no fever, but in weakly persons.
- (5) Early cases, "irritative," such as follow influenza.
- (6) "Catarrhal phthisis," i.e. with much bronchial catarrh.
- (7) So-called "scrofulous phthisis," i.e. with other tuberculous lesions in glands, bones, etc.
- (8) Fibroid phthisis.
- (9) Bronchiectatic phthisis.
- (10) Phthisis with emphysema.
- (11) Laryngeal phthisis.
- (12) Elderly patients.
- (13) So-called "erethismic" patients, i.e. "nervous irritable subjects, neuralgic, dyspeptic, bad sleepers, with habitually quick pulses and dry, harsh skins."
- (14) Anæmic patients.
- (15) Patients with heart disease, arterial degeneration, feeble circulation, "chilly people."

(1) Early cases, young, vigorous, uncomplicated, with little or no fever, should probably be sent to the Swiss Alps in preference to any other locality; and of the Alps we may safely take Davos as a type. Their chance of recovery in suitable Alpine stations is probably greater than elsewhere. There is rail all the way to Davos, and the journey from London takes about twenty-four hours; it is usually

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unnecessary to break the ascent. The end of September is the best time to arrive, and the patient should be prepared to remain two winters and a summer. He may, however, go to Davos later in the season, though better not so late as the end of January. On returning, the descent may with advantage be broken, and Ragatz or Wesen may be chosen as a halting-place.

If some other station than Davos is proposed, the conditions which seem important in the Davos climate should be remembered in discussing it. These are—(a) Low barometric pressure—about 5 in. below that of sea-level; (b) dryness of air; (c) purity of air; (d) coldness of air; (e) diaphaneity and diathermancy of air; (f) air-stillness. This air-stillness has probably received far too little attention; when the snow has fallen at Davos the stillness is remarkable, contrasting with what is found in some other high-altitude stations which have given less satisfactory results.

Here also it is necessary to recall the sort of cases which should not be sent to the high Alps, because, whilst powerful for good in suitable instances, these altitudes are powerful for harm in unsuitable cases. Such are—(a) Cases of recurrent hæmorrhage from a cavity; (b) the acute stage of phthisis in any form; (c) the "erethric" constitution at any stage; (d) laryngeal cases as a rule; (e) advanced cases; (f) cases with marked emphysema; (g) cases with albuminuria; (h) cases with intestinal irritation; (i) cases with general bronchial irritation; (j) elderly persons; (k) persons who cannot stand cold easily.

(2) In early cases with recurrent hæmorrhages, after some weeks have elapsed since the last hæmorrhage (weeks which may have been well spent in a sanatorium), the Alps may be advised.

(3) Early cases which have originated in pleurisy or pneumonia should also be sent to the Alps.

(4) Early cases, uncomplicated, with little or no fever, in weakly persons, *if they do not feel cold easily*, may also do well at Davos. But usually a warmer climate will have to be recommended. If sent to Davos, they should break the ascent at some intermediate altitude, such as Ragatz (1,700 ft.) or Wesen (1,425 ft.). If Davos be decided against, they may be sent to Egypt, the Riviera, or to a sanatorium. The season in Egypt is an exceptionally short one; patients arrive in

November and must generally leave in March: the journey takes about six days from London and three of these are spent at sea; Helouan near Cairo, Luxor, or Assouan may be chosen; patients leaving Egypt must find some other resting-place until the end of May at earliest; probably the Riviera provides the safest. The expense of Egypt is considerable, and patients subject to diarrhoea should not be sent there. The Riviera is nearer home and less expensive; patients should arrive about the last week in October and not return to England until the end of May; Nice is to be avoided; Cannes is uncertain; Hyères is very windy in February and March; San Remo and Mentone are probably among the best places on the coast. Dr. Samways, of Mentone, has seen some excellent results, and points out that the climate is by no means a "marine climate" in the usual sense, since the winds are so predominantly from the mountains, rendering the air dry and bracing. Nervous, "highly-strung" persons and bad sleepers, however, may find the Riviera too "exciting" for them.

With regard to *sanatoria* at home and abroad, it is still very difficult to make comparisons. The statistics are not yet easily comparable, and enthusiasm seems sometimes to lead to over-statement. So far as I can observe, those sanatoria placed with due regard to the avoidance of the unfavourable conditions enumerated at the beginning of this section (p. 255) have attained the greatest measure of success. But a careful investigation is required. The statistics of the German sanatoria published in 1909 show that their results were far from uniform, and although, with the information given, it is impossible to reach really definite conclusions as to the causes of the differences, the differing climates of the districts dealt with may plausibly be held to be in part responsible.

Attention must here be drawn to some remarkable figures published by Dr. Saugman from the Vejle fjord sanatorium in Denmark. This institution stands near the northern shore of the Vejle fjord on the Danish east coast, apparently in considerable shelter. The results are said to be as favourable as those obtained at Davos; they are also said to be unusually permanent. One cannot but remember, in considering possible causes for this, the fact that in Denmark unique measures have been taken to secure for the population a tubercle-free milk supply.

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(5) Post-influenzal cases with considerable irritation are said to do particularly well on the Riviera.

(6) "Catarrhal phthisis" is the one variety of the disease said to do well in Madeira. But Madeira has a risky climate, and it may probably be wiser to send the patients to sheltered parts of Torquay or Sidmouth. Cases with a tendency to diarrhoea must avoid Madeira.

(7) Patients who are the subjects of "scrofulous" phthisis may spend the winter on the Riviera and the summer at sheltered sanatoria in the eastern and south-eastern English counties.

(8) Sufferers from fibroid phthisis will probably do as well on the Riviera as anywhere.

(9) Cases of bronchiectatic phthisis perhaps do best in Egypt.

(10) In phthisis with emphysema the patients progress satisfactorily on the Riviera or in Egypt.

(11) Patients suffering from laryngeal phthisis may do well at Mentone or San Remo, or under skilled treatment in a well-placed sheltered sanatorium.

(12) Elderly patients will find the Riviera or Egypt their best wintering place.

(13) "Erethismic" patients find that Egypt suits them best.

(14, 15) Anæmic patients, and patients with heart disease, arterial degeneration, etc., should probably choose the Riviera or Egypt.

It will be observed that a very limited list of localities for phthisis cases has here been given. By thus confining attention to a few places, needless complexity is avoided and only resorts with well-established reputations are cited. There are, however, other foreign resorts either possessing unquestioned merit or at least so well known that patients will inquire about them. The chief are as follows: The high Andean valleys of Peru *perhaps* offer the finest climates for phthisis in the world, and now that good railways and steamers are available and the isthmus of Panama is shorn of its malaria and yellow fever, a fresh opportunity of using them arises. Jauja can be reached from London in little over a month. Whoever goes, however, should be prepared to rough it. South Africa has, in its high tablelands, climates of undoubted value. But accommodation is limited, living dear, and employment not easy to get. The coast must be avoided. Ceres, Middelburg, Craddock, and Aliwal North are among the best-known resorts. Natal is not to be recommended.

North America, Australia, and New Zealand have in operation laws relating to phthisical would-be immigrants which had better be studied before advising patients to go to them. The Canaries have climates which are very diverse. Las Palmas, in Grand Canary, is well deserving of attention. It is dry and sunny with little wind or rain. To class it with Madeira is a mistake.

Sea voyages can no longer be safely advised, except as a means of conveyance to other countries. They have proved curative, it is true, in many instances, but the percentage of success has been less than on land, and in bad weather the hardships are considerable, especially for women.

Home climates for phthisis.—Whether for rich or for poor, the majority of cases will probably continue to be treated in this country. Therefore it is the more regrettable that we cannot say definitely that, for such and such a type of case, this or that locality is best. Until reliable statistics are forthcoming of sanatorium results, reasonably comparable, for a period of, say, ten years, from different parts of the country, knowledge will remain thus imperfect.

At the same time, it is possible to say that the desirable conditions for a consumptive's place of residence, both during treatment *and after*, are a dry soil, pure, dustless, fogless air, abundant sunshine, protection from wind, especially from rainy wind, and a position at least a quarter of a mile inland from the sea. It is probably worth while to choose a district in which phthisis mortality is low, and where good reason exists for believing that this low mortality is at least partly due to climatic factors.

Besides these general rules, some local and seasonal indications may be added. For the more vigorous patients, and especially in summer, the east and north-east of our islands are perhaps best. Such places as Nairn, Braemar, and the Dee Valley, Kingussie, the East Anglian and Kentish coasts, may be specially pointed out. But Hastings, the north-east of Dartmoor, and Malvern also deserve mention.

In winter and spring, and generally for the less robust, the southern counties of England furnish satisfactory conditions. Sheltered parts of Penzance, Paignton, Torquay, Sidmouth, Ventnor, Bournemouth, and dry sunny valleys in their vicinity, afford considerable choice.

3. *Phthisis patients who must decide for*

CLUBBED FINGERS AND TOES

themselves whether or not to remain at home.—These may be classified as—(1) patients with early phthisis and some albuminuria, (2) patients with advanced phthisis, (3) patients with recurrent hæmorrhage from a cavity.

Practical difficulty arises in these cases chiefly with regard to foreign resorts. We may therefore content ourselves with a brief recommendation of places abroad.

(1) Patients with early phthisis and some albuminuria had better choose the Riviera or Egypt.

(2) Those with advanced phthisis had better let the Riviera be their limit of distance.

(3) Those with recurrent hæmorrhage from a cavity will be safest in some sanatorium.

Pneumonia (chronic).—The high Alps are best for the young, Egypt and the Riviera for the elderly.

"Rheumatism" (chronic), including the various complaints commonly so called, requires a warm, dry climate. Helouan, near Cairo, probably owes its usefulness to its climate as well as to its baths. Certain sea-side places do not agree.

Scrofula is generally best treated at the seaside all the year round. The east and south-east coasts of England possess admirable climates. The coast of Thanet, at Margate especially, enjoys a well-earned reputation.

Skin diseases (chronic).—Mere change of air and scene, with rest, may benefit chronic urticaria. Eczema is usually made worse by damp, cold, and wind; also, at first at least, by sea-air.

WILLIAM GORDON.

CLINICAL BACTERIOLOGY AND PATHOLOGY (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

CLUBBED FINGERS AND TOES.—This term implies an enlargement of the terminal phalanges of the fingers and toes. The nails are curved laterally and longitudinally, and are sometimes spoken of as "filbert-shaped." The condition occurs in chronic heart or lung disease. In the former, it is especially marked in congenital affections; in the latter, in bronchiectasis, fibroid phthisis, and other forms of fibrosis, old-standing empyema, pleuritic adhesions, and emphysema. It is generally associated with chronic cyanosis. Pulmonary osteo-arthritis may also be present. In hepatic cirrhosis, especially in children, clubbed fingers and toes are occasionally met with. Hanot's biliary cirrhosis is the type most often

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accompanied by this abnormality. A particular form of familial and hereditary clubbing unaccompanied by symptoms has been described. Opinions are divided as to whether digital clubbing is due to chronic venous congestion or to the local effects of toxins. It is usually persistent, but may be transitory.

FREDERICK LANGMEAD.

CLUB-FOOT (see TALIPES).

COCAINE HABIT (see DRUG HABIT).

COCAINE POISONING (see POISONS AND POISONING).

COCYGOODYNIA (see NEURALGIA).

OCELIA DISEASE (see MORBUS CÆLIACUS).

COITUS, PAINFUL OR DIFFICULT (see DYSPARUNIA).

COLD, COMMON (see CORYZA).

COLIC.—A severe abdominal pain occurring in paroxysms of short duration. The forms usually recognized are renal, biliary, and intestinal, renal and biliary colic being due to disordered peristalsis of the ureter and bile-duct respectively, usually in association with the passage of a calculus. In renal colic the obstruction in the ureter may be due to kinking, either through hypermobility of the kidney or over an aberrant renal vessel. Pancreatic colic, which results from the passage of a pancreatic calculus, is rare. Pain resembling colic to some extent may be provoked by the abdominal crises—gastric, intestinal, or ureteric—of tabes, or may be due to angina abdominis or be referred to the epigastrium in angina pectoris. *Intestinal colic* owns many causes, but occurs more particularly in inflammatory conditions of the bowel, such as enteritis, dysentery, and appendicitis, or when within the bowel are irritating contents such as indigestible or decomposing food, unripe or overripe fruit, purgatives, irritant poisons, or bacterial toxins as in summer diarrhoea. Flatulence is sometimes a sufficient cause, especially in infants. Lead-poisoning provides a special form. Both nervous diarrhoea and spasmodic constipation are sometimes accompanied by colic; in the former a hurried peristalsis occurs immediately after the taking of food, and severe abdominal pain, colicky in nature, may immediately be complained of, and be relieved by the precipitate passage of a stool. The relationship

of cold and heat to attacks of colic is well defined but difficult to explain. In some patients a sudden cold spell heralds with great regularity abdominal pain of this nature, whilst others react in the same manner to a rapid rise of atmospheric temperature.

It is in the nature of colic to be sudden and severe, doubling the patient up in its intensity, causing nausea and sweating and sometimes pallor, a quick, feeble pulse, or even syncope. Infants may become blue, semi-conscious, or convulsed. Collapse may follow repeated attacks. Abdominal tenderness is often absent; it may be obvious in severer attacks, especially if they are repeated, but does not amount to that which accompanies peritonitis.

The **diagnosis** of colic is dealt with in the article on ABDOMINAL PAIN, DIAGNOSIS OF, and the several forms are described in detail under their appropriate heads (see URINARY CALCULI; GALL-STONES; APPENDICITIS, etc.).

Treatment.—The treatment of intestinal colic as a symptom may be considered here, though a warning must be given at the outset against the symptomatic treatment of colic while its cause is still in doubt. This applies especially when a suspicion of appendicitis or other disease requiring surgical treatment arises, for the use of opiates—the drugs most likely to give immediate relief—may mask the symptoms and lead to dangerous or fatal delay, while purgation may be equally inimical.

If the colic is not accompanied by diarrhoea, it is a wise procedure to begin treatment by the administration of a large soap-and-water enema; this may bring away hard and scybalous masses and lead to the passage of flatus, the colic being thereby ended. If the colic continues, or if diarrhoea is present, castor oil ($\frac{1}{2}$ oz.) combined with 10 min. of laudanum or liq. opii sedativus will often prove successful, and at the same time rid the bowel of its disturbing contents. Opium in one or other of its forms is unquestionably the surest means of allaying the pain, and of these none is more efficacious than chlorodyne (tinct. chlorof. et morph. co.) in 7- to 10-min. doses. If the offending material remain in the bowel, however, the colic is likely to appear again. For this reason, opium should if possible be deferred until free purgation has been obtained, unless it be combined with castor oil.

Heat applied to the abdominal wall often relieves; it may be in the form of hot bottles, fomentations, poultices, or a mustard plaster.

The same is true of gentle massage with the palm of the hand anointed with warm olive oil, a method especially useful in infants. The colic of "nervous diarrhoea" may be prevented in many cases if meals be taken regularly and slowly, very hot or cold food avoided, and small doses of potassium bromide given regularly thrice daily. A valuable prophylactic measure for those who suffer from colic with sudden heat or cold is the so-called "cholera belt."

FREDERICK LANGMEAD.

COLITIS.—Clinically, the varieties of colitis met with fall into three groups, though numerous minor forms are encountered. They are—

1. Simple colitis.
2. Mucous colitis.
3. Ulcerative colitis.

1. SIMPLE COLITIS

This form of colitis consists in a catarrhal inflammation of the mucous lining of the whole or a part of the colon. The disease may be primary, or may occur as a complication of some other disease, such as nephritis, diabetes, pneumonia, etc. It usually starts acutely, but if untreated may become subacute or even chronic. It may follow some dietetic indiscretion, exposure to cold, or the use of violent purgatives.

Symptomatology.—The symptoms vary much in severity. In moderately severe cases there are abdominal pain and tenderness, often most pronounced along the course of the colon or a part of it, such as the sigmoid flexure. Diarrhoea occurs, the motions consisting sometimes of mucus mixed with blood. The temperature is often raised, and there may be febrile symptoms. In severe cases there may be profuse hæmorrhagic diarrhoea and collapse, whilst, on the other hand, it is probable that some cases of transient colicky diarrhoea are mild examples of a combined entero-colitis.

Treatment consists in rest in bed, liquid diet, the application of hot stupes and poultices to the abdomen, and the administration of sedatives such as bismuth salicylate in combination with nepenthe or tincture of opium. If there is much pain a starch-and-opium enema or a morphia suppository should be employed.

2. MUCOUS COLITIS

Mucous colitis, known also as Muco-membranous Enterocolitis, Membranous Catarrh of the Intestines, Tubular Diarrhoea, is characterized by the passage of mucus from the bowels,

the occurrence of attacks of abdominal pain, often severe, and the presence of various digestive disturbances.

Etiology and pathology.—The condition is more common in women than in men, and is met with principally between 25 and 45 years of age. A majority of the cases present marked neurotic symptoms, and this, coupled with the fact that in some cases no pathological lesion has been found in the colon, gave rise to the view that the disease was essentially one of the nervous system; but more recently it has been recognized that in most cases some pathological lesion is present and is the essential cause. According to Mummery, the term mucous colitis denotes "a collection of symptoms, which may be due to many different pathological conditions of the colon, of widely different characters." Among these conditions are (a) *chronic catarrhal colitis*; (b) *chronic appendicitis*: in many cases it is probable that appendicular inflammation is primary, and colitis secondary, due either to the passage of inflammatory products from the appendix into the colon or to adhesions; (c) *peritoneal adhesions*: these are not an uncommon cause and may follow operations, or be secondary to peritonitis, or to disease of pelvic organs; (d) such rarer causes as enteropositis, intestinal parasites, and carcinoma of the colon. Much importance must be attached to chronic constipation, and in the opinion of von Noorden it is such a potent factor that the colitis will be cured as soon as the constipation is got rid of. Irritation by powerful purgatives, or by the products of bacteria from a septic mouth or elsewhere, may also play a part.

Symptomatology.—The most characteristic symptom is the passage of mucus per anum. A little mucus in the motions may have been noticed for years before other symptoms develop, more especially if constipation exists. As the disease advances it becomes more abundant, and may be passed as soft glairy material, alone or mixed with faeces. Sometimes abundant mucus of this form persists more or less permanently for years, and, together with general abdominal discomfort, loss of weight, anæmia, and general ill-health, constitutes the entire symptomatology. More often the mucus appears at times only, in the form of shreds and membranes, which may prove to be casts of the bowel; even solid cylindrical masses, a foot or more in length, are passed occasionally. The membranes are

often tough and even fibrinous in appearance, but they are composed solely of mucus with a few desquamated epithelial cells. The appearance of mucus in the membranous form is usually accompanied by spasms of abdominal pain which constitute the second great feature of the affection. It varies in severity, sometimes being merely an exaggerated form of the habitual abdominal discomfort, and at others so severe as to produce profound collapse, and even to simulate an acute abdominal catastrophe. All grades of attacks between mild and severe occur, those of moderate severity being most frequently met with. They are caused by the violent peristaltic unrest of the colon directed to the separation and expulsion of the membranes which are closely adherent to its surface. The third great symptom is irregularity of the bowels. In almost all cases there is a history of long-standing constipation, possibly alternating with occasional severe diarrhoea. Inquiry will generally show that, even when diarrhoea has been present, hard scybala have been passed with the liquid motion, which points to the irritation of these masses as the cause of the diarrhoea. Habitual intestinal discomfort is seldom absent; it is variable in character and in its time of onset, and worrying in nature. The general health suffers, and, together with the anæmia and emaciation which develop, there is frequently pronounced neurasthenia. The patients' symptoms come to dominate their entire existence. Peevishness and irritability follow, and a picture of neurotic invalidism in the most pronounced form is finally produced. In women some form of uterine trouble is usual.

Physical examination of the abdomen reveals nothing characteristic, but evidence may be found of one or more of the pathological conditions already referred to as possible causes. Alternate dilatation and constriction of the colon, and especially of the cæcum, may be felt.

In a certain proportion of cases, usually of the severe type, intestinal sand of a gritty nature and reddish colour may be evacuated. Its passage is usually accompanied by considerable pain and some bleeding. The exact significance of this sand is not understood. It is undoubtedly met with independently of mucous colitis. Analysis shows that it consists of the phosphates of calcium and magnesium, and of about 50 per cent. of organic matter, with traces of urobilin.

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Diagnosis.—This is seldom difficult, though it will frequently be impossible to determine the exact pathological condition underlying any given case, and some associated condition such as uterine disease, or nephroptosis, may overshadow the symptoms of colitis, especially in the earlier stages, and appear to be the primary or sole morbid condition present. *Tuberculous peritonitis*, particularly its adhesive form, though not very common in adults, may closely simulate mucous colitis. Much mucus may be passed, and there may be attacks of colicky pain caused by pressure of the adhesions on the intestinal coils. As a rule, however, a careful abdominal examination, and the existence of symptoms pointing to tuberculous disease, such as a rise of temperature, will enable a diagnosis to be made. The acute attacks of spasmodic pain may simulate *intestinal obstruction*, or even the *perforation of a hollow viscus*. The history of previous attacks, the passage of mucus, and a careful examination should suffice for the avoidance of this error.

Prognosis.—If a case is recognized early and is at once submitted to appropriate treatment, a favourable result may be anticipated within six months or a year. Old-established and severe cases are very intractable, though uncomplicated mucous colitis never proves fatal. It is common to meet with cases that have persisted for ten years or more, during which there have generally been periods of comparative health followed by fresh exacerbations. Prolonged and careful treatment usually ameliorates even the worst cases.

Treatment.—First, it is important to estimate the effect of any pathological condition present, and to treat it. For example, if there is sufficient evidence to show that chronic appendicitis is the cause, appendicectomy should be performed, and a complete cure may result, though more frequently there is only improvement and further treatment is required. In other cases peritoneal adhesions or enteroptosis may be present, and must be dealt with, the former by operation, the latter by the use of supporting belts. The treatment of the acute attacks of pain consists in absolute rest, the administration of a hypodermic injection of morphia or of omnopon, and hot applications to the abdomen. A suppository containing extract of belladonna ($\frac{1}{4}$ – $\frac{1}{2}$ gr.) may also prove useful, and, when the pain becomes less acute, a hot soap-and-water

enema, or an injection of half a pint of warm olive oil. The enema usually brings away most of the irritating mucus, and is followed by a period of quiet sleep.

Diet.—When they commence systematic treatment, many patients are thin and emaciated not merely from their disease, but from having voluntarily cut down their diet in the belief that one article of food after another is responsible for their suffering. For them, undoubtedly, a more generous diet is required in order to increase their strength and resisting power, but few English patients can tolerate the bulky diet advocated by Langenhagen. It is wise, indeed, to supplement the diet gradually. A moderate amount of cream, butter, and bacon fat may be given; junket and milk, if liked by the patient, may be taken in abundance; if ordinary milk disagrees, butter-milk or Bulgarian milk may be tried. In many cases the farinaceous soups advocated by Combe of Lausanne are most useful; they can be made with rice, oatmeal, barley, tapioca, lentil flour, etc., and either with milk or with water; the special Swiss preparations are now obtainable in England. Eggs, more especially the yolks, may be taken, and, provided there is no evidence of great intestinal decomposition, fish, chicken, and tender meat. If there is much anæmia, raw-meat juice may be ordered. Stale white bread toasted and rusks soaked in milk are allowable. Raw vegetables, fruit, and all obviously indigestible substances should be avoided at first. Among beverages, milk, weak tea, cocoa, and saline waters such as those of Kissingen and Homburg, are the best. Thorough mastication, and rest, both before and after meals, should be enjoined.

Even more important than diet is the maintenance of a *regular action of the bowels*. Powerful aperients are to be avoided. In many cases liquid paraffin, $\frac{1}{2}$ oz. twice or three times daily, will prove most satisfactory; but if more is required a small dose of castor oil may be given each morning, or a teaspoonful of the old-fashioned senna prune conserve. A few patients find that a saline draught in the morning agrees best, and is less unpalatable than either paraffin or castor oil, but it is usually less efficacious. "Regulin," a preparation of cascara and of agar-agar, and "thaolaxine," a somewhat similar compound, are also useful; their action, like that of liquid paraffin, is largely mechanical. At the beginning of a course of treatment, olive oil enemata are most useful; 10–15 oz. of warm

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oil should be injected slowly into the bowel at night, and if possible retained till morning. The oil helps to soften hard scybala, and probably also acts as an emollient to the colon.

In cases where pain is not too severe, *massage* of the colon is useful. It must be commenced in the most careful manner, as it is impossible at first to foretell if it will irritate the bowel. General massage also is of use in improving the general nervous tone.

Colonic *lavage* is carried out systematically at many spas, and, carefully performed, is beneficial if a limited course only is prescribed. If the patient cannot go to a special resort, the lavage may be done at home, plain warm saline being employed, or some simple astringent such as borax (30 gr. to the pint), or solution of protargol ($\frac{1}{2}$ per cent.). In no case should the lavage be carried out for a longer period than three weeks. Unnecessary and prolonged washing out of the colon is likely of itself to produce catarrhal colitis.

Vaccines.—Attempts have been made to treat this disease by means of vaccines. The organism usually found is a variety of the colon bacillus, but occasionally streptococci are present in the mucus in abundance. Unless actual inflammation of the colon is the main pathological change, it is unlikely that vaccines will prove of much use, but in conjunction with other measures they are worth a trial; some cases appear to be benefited.

Bulgarian milk agrees well with many patients, and undoubtedly the intestinal flora may be modified by its use. The exaggerated claims made for it have not been substantiated by experience. Tonics may be given, and every step should be taken to improve the general health. Fresh air and moderate exercise are obviously beneficial. Formerly intestinal antiseptics were much recommended, but they are of little or no use. The best means of preventing excessive intestinal putrefaction consists in ensuring thorough and regular evacuation.

Surgical treatment.—In some cases, in spite of every care, and the most scrupulous attention to medical treatment, the patient remains a chronic invalid, and life is rendered almost intolerable by the extreme abdominal discomfort and the frequent passage of mucus. For these surgical treatment should be considered. The best operation is excision of the colon; it is, however, a very serious procedure, and should never be undertaken until everything else has failed, and after the patient has been fully informed of the risks. Appendicostomy is

often disappointing in its results. The operation has the merit of being comparatively safe, and is easy to perform. Many, therefore, will like to try this procedure before having recourse to the more serious step of colectomy. Other operations are cæcostomy and ileo-sigmoidostomy. The former, even in favourable circumstances, may leave the patient in a most uncomfortable condition from the passage of fluid fæces on to the abdominal wall; the latter is by no means free from danger, and the short-circuited colon may become filled with regurgitated fæces.

3. ULCERATIVE COLITIS

Ulceration of the colon is not uncommon, and the following varieties are recognized, viz. (a) *follicular ulceration*, which is usually secondary and is rarely recognizable clinically, though diarrhoea may be present as a symptom; (b) *stercoral ulceration*; (c) *vascular ulceration*, which is the result of either venous or arterial disease, and is met with in cirrhosis of the liver, and in cases of embolism of the mesenteric arteries; (d) *trophic ulceration*, occurring in the course of diseases of the nervous system, and usually accompanied by the passage of bloodstained mucus from the bowel; (e) the ulcerative colitis of *enteric fever*, of *tuberculous enteritis*, and of *syphilis*; (f) *malignant ulceration*.

There exists also a group of diseases characterized pathologically by ulceration, more or less diffuse, of the colon, and by general catarrhal colitis, and accompanied by distinctive clinical features. This group includes (a) *amæbic dysentery*, (b) *bacillary dysentery*, and (c) *ulcerative colitis proper*. Much difference of opinion exists as to whether bacillary dysentery and ulcerative colitis are identical diseases, but in any case it is important to recognize that an acute and serious form of ulcerative colitis does occur sporadically in this country. This condition is the only form of ulceration considered in this article. (See also DYSENTERY.)

Etiology and pathology.—Ulcerative colitis is most frequent in adult life, and equally common in males and females. Nothing is known of its etiology, but it is probable that prolonged constipation is a predisposing cause. Pathologically, intense inflammation of the mucous lining of the colon exists throughout the whole or part of its extent. The degree and extent of the ulceration is most variable. Sometimes only scattered ulcers exist, whilst

in other cases whole tracts of the colon may be denuded of their mucous membrane, save for shreds persisting here and there and resembling polypi owing to the depth of the adjoining ulceration. Perforation is met with comparatively frequently. In cases of long duration the wall of the colon may be thickened and leathery in appearance, due to fibrous changes in its wall.

Symptomatology.—The most important symptom is diarrhoea. It may start suddenly or gradually, but, when the disease is fully developed, from six to twelve motions daily may be expected. The motions are fetid, dark-coloured and fluid, and contain altered mucus and blood. Shreds and sloughs of mucous membrane may also be seen, and, microscopically, collections of pus cells. Attacks of gripping abdominal pain precede or accompany the motions, and as a rule some tenderness is found on pressure over the colon. The patient soon develops the general symptoms of severe illness. There are wasting, anæmia, loss of appetite, irregular temperature, sweating, and vomiting; according to some writers, hiccough is a common symptom. Unlike tropical dysentery, tenesmus is not often complained of, and as a rule the motions are more faecal in character than in dysentery. The most common complicating disease is interstitial nephritis.

Diagnosis.—This is seldom difficult, and if a sigmoidoscope is employed no doubt should exist in differentiating the disease. With this instrument, which must be used with extreme care, the swollen congested mucosa may be seen, and actual ulcers may be detected. In cases which start very acutely, *intussusception* may be suspected, but the absence of obstructive symptoms, and the fact that *intussusception* is most frequent in childhood, should eliminate this possibility. *Malignant disease* of the colon may also give rise to difficulty, but its symptoms seldom set in so acutely.

Prognosis.—Ulcerative colitis has a high mortality. The duration of the disease varies from a few days to as many years, but "the patient is usually dead within eight weeks from the commencement of the illness" (Hale White). However, a chronic variety with occasional acute exacerbations is not infrequent.

Treatment.—Hitherto treatment has met with but scant success. Of late many surgical procedures have been advocated. These include appendicostomy, cæcostomy, colostomy, and ileo-sigmoidostomy. In very acute cases

probably the best course is to perform an immediate colostomy, whilst in less acute cases medical treatment may be tried, but if it fails surgical intervention should not be long delayed. Medical treatment includes complete rest, careful dieting with articles of food that leave little residue, such as milk, eggs, jelly, milk puddings, and chicken tea, and the application of hot stupes or poultices to the abdomen. The pain and diarrhoea must be relieved by the free administration of opium, with which may be combined bismuth salicylate. Some writers strongly advocate the free use of calomel in the earlier stages of the disease. Washing out of the bowel is often too painful to be borne, but may be attempted with plain saline solution or a weak solution of argyrol (1 per cent.). If hæmorrhage is severe, tincture of hamamelis, 2 dr. to 1 pint, or diluted adrenalin, 1 dr. to 1 pint, may be used in the douches, and in addition fresh serum (10–20 c.c.) should be injected hypodermically. Neither intestinal antiseptics nor artificially soured milk are of apparent value.

Recently, *vaccines* have been employed. The organism most commonly met with is the *B. coli communis* apparently in an unusually virulent state. Streptococci also occur. Improvement certainly follows the use of vaccines in some cases, but relapse is likely to occur, and it is advisable when this treatment meets with success to persist in it, using increasing doses even after the patient seems perfectly recovered. The vaccine should always be an autogenous one. Anti-colon-bacillus serum has so far not proved valuable, but some success has been claimed for the anti-dysenteric serum prepared by the Lister Institute.

T. G. MOORHEAD.

COLLAPSE (see SHOCK AND COLLAPSE).

COLLAPSE OF LUNG (see LUNG, COLLAPSE OF).

COLLOIDS. *Chemistry of colloids.*—The earliest examination of the properties of colloids was that of Graham in 1861. Graham distinguished colloids from crystalloids, the former being substances which crystallize with difficulty and which in a solvent do not dialyse through a parchment membrane, and the latter substances which are readily crystallizable and dialyse. We now know that many crystallizable substances form colloidal solutions under appropriate conditions, and, in speaking of colloids, we refer only to a definite physical state of matter into which probably all chemical

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substances can be brought by the use of suitable chemical or physical methods. Graham termed a colloidal solution a "sol," and gave the name "gel" to the jelly-like mass produced by the coagulation of a sol. Colloidal chemistry is now a subject of large dimensions, and it is impossible in small space to do more than indicate the properties of matter in the colloidal state and outline briefly some of the parts played by colloids in the animal organism and in therapeutics. References are provided to sources where further information may be obtained.

The distinction between true solutions, colloidal solutions, and suspensions depends on the degree of fineness or dispersion of the dissolved substance in the solvent. A *true solution*—i.e. where the solute is at least in a state of molecular dispersion—appears, even in the ultramicroscope, optically homogeneous; and it dialyses and diffuses readily. A *colloidal solution* is optically often turbid and in the ultramicroscope is heterogeneous, since it exhibits the Tyndall cone of light owing to diffraction from its suspended particles; it does not dialyse or diffuse readily, but its components are not separable by ordinary filtration or by gravity. A *suspension* or *emulsion* is optically heterogeneous to the naked eye, it does not dialyse or diffuse, and its components are separable by filtration or by gravity on standing. By the use of suitable dialysing membranes and of pressure, colloids may be subjected to ultrafiltration. From the size of the pores in these membranes the sizes of colloidal particles have been found to lie between $200\ \mu\mu$ and $20\ \mu\mu$ or even less ($\mu\mu$ = one-millionth millimetre), and this has been confirmed by the Tyndall phenomenon, since the size of particles which will so diffract light must be less than half the wave-length of the visible rays.

The two phases of a colloidal system may be variously solid, liquid, or gaseous, and we may have, as types of colloidal states, water of crystallization (liquid in solid), suspensions (solid in liquid), emulsions (liquid in liquid), tobacco smoke (solid in gas), fog (liquid in gas), foams (gas in liquid), etc. In colloidal chemistry the dissolved substance (solute) is termed variously the "internal phase" or "disperse phase," and the dissolving substance (solvent) is termed the "external phase" or "continuous phase." The same substance may form at one time an internal phase and at another an external phase; thus we may have a colloidal oil in which the oil is the internal phase in an

external phase of water and, vice versa, a colloidal water in an external phase of oil; the former condition may be illustrated by cream, the latter by butter.

Colloidal solutions are divisible into suspension colloids and emulsion colloids. In **suspension colloids** (suspensoid or lyophobic colloids) the viscosity is practically the same as that of the continuous phase, and they are readily precipitated by the addition of low-percentage salt solutions. To the suspensoids belong the metallic sols in general, and it is assumed that in these the disperse phase is in the form of solid particles which are aggregates of molecules. **Emulsion colloids** (emulsoids or lyophilic colloids) possess a viscosity higher than that of the continuous phase, and are precipitated only by high-percentage salt solutions. To the emulsoids belong the organic colloids, e.g. proteins, gelatin, gums, starch, soaps, etc., and in them the disperse phase is liquid. The two liquids forming an emulsoid system are not necessarily entirely discrete; in an albumin sol, for example, there is a continuous phase of a dilute solution of albumin in which are suspended minute globules (disperse phase) of a more concentrated albuminous solution, i.e. the two phases contain both components in different concentrations. The precipitation or "salting out" of emulsoids by high-percentage salt solutions, e.g. NaCl , Am_2SO_4 , etc., is due first to the removal of water by the salt from the disperse phase to the continuous phase, with the result that the disperse phase becomes more solid and resembles a suspensoid particle, so that it is precipitated. In many cases a reversal can take place and the precipitate on removal of the electrolyte redissolves. Weak solutions of albumin, when boiled, show only opalescence, but, on the addition of an acid or electrolyte, flocculation takes place by agglomeration of the particles, just as mist forms rain. The formation of gels is commonly found in emulsoids. Thus a strong solution of gelatin on cooling sets to a jelly which behaves much like a solid and resists, even under strong compression, removal of its contained water. Its sol conditions have, therefore, been reversed, and the water has now become the disperse phase and exists as globules surrounded by a solid sponge-like continuous phase of gelatin. Many gelations are reversible, but silicic acid sets to a gel which is irreversible. Many of the organic emulsoids when dried exhibit the phenomenon of imbibition, e.g. gelatin.

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Positive and negative colloids.—The division of colloids into positive and negative has more particular reference to the behaviour of suspensoids, which are readily thrown out of solution by electrolytes. Most substances in colloidal solution with water assume an electric charge, which is situated at the boundary surface between the two phases and originates in most cases from electrolytic dissociation. Thus, a colloidal particle of silicic acid in water consists of an aggregate of molecules composed of silicate anions (—) and hydrogen cations (+); in this case, the more mobile hydrogen cations will migrate, leaving the particle as a multivalent anion. With basic colloids, e.g. aluminium hydroxide, OH anions will migrate and leave a multivalent cation. The migrating ions are, however, kept in close approximation with the less mobile ones by electrical attraction, hence they cannot be removed by dialysis.

The electrical charge of a colloidal particle may be estimated (a) by its migration in an electric field—electrophoresis—a negative colloidal particle travels towards the anode; (b) by precipitation with a known oppositely charged colloid—ferric hydroxide is a positive and arsenious sulphide a negative colloid; (c) by capillary analysis with filter paper which has a negative charge. The electrical charge can be influenced by electrolytes; thus a suspensoid particle having little electric charge may in acids assume a positive charge and in alkalis a negative charge. Alteration of the electrical charge of suspensoid particles produces agglutination, aggregation, and precipitation of the particles, and, if a salt be employed for this purpose, it is the ion having the opposite electrical charge to the colloidal particle which is the precipitant. The power of such ions as precipitants increases in proportion to their valency. The mechanism of the precipitant action of weak solutions of electrolytes on suspensoids is thus connected with the neutralization of the charge on the suspensoid particles, and is an adsorption effect. The electric condition of the emulsoids and of inert insoluble particles is determined almost entirely by the reaction of the dispersion medium, i.e. the proportion in it of H and OH ions. Albumin, free from electrolytes, appears to have no charge, in acid media it assumes a positive charge, in alkalis a negative charge. While emulsoids are not generally sensitive to weak solutions of electrolytes, the difference between them and suspensoids in this respect is not fundamental but is one of degree. In the

phenomenon of heat coagulation, e.g. of proteins, the emulsoid first changes in character to resemble a suspensoid in that it becomes sensitive to electrolytes; secondly, agglutination of the colloid particles follows as a result of the action of the electrolytes.

The permanency of colloidal solutions depends on various factors. (1) Deposition is delayed by the small size of the particles, on which gravity, as against the viscosity of the external phase, has little effect. (2) The density of the external phase, if it is high, will increase the permanency. (3) Examination shows the particles to be in active motion—Brownian movement—which is the resultant of the impacts of the molecules in the external phase on the colloid particles. (4) The particles, having similar electrical charges, mutually repel one another.

Surface tension and adsorption.—Colloidal systems exhibit very strongly the phenomena of surface tension and energy which are evolved at the surfaces of contact between the internal and external phases. Surface energy is proportional to the product of the surface tension and the surface area; hence in a colloidal system where the internal phase is in a state of high dispersion (i.e. large surface with small volume) the internal energy represented is considerable, and, in the case of a liquid disperse phase, the internal component of the surface tension is great enough to confer a solid-like rigidity on the particle. It is a well-known law that free energy is always reduced to a minimum, and this applies to the surface energy in a colloidal system. One factor, the surface area, is reduced to a minimum by the assumption of the smallest surface area (i.e. spherical shape) by the internal phase; surface tension, the second factor, can be reduced in a fluid by the addition of soluble substances—a few inorganic salts, however, raise surface tension—and it has been found that, in order to achieve the maximum reduction of surface tension, the molecules of the dissolved substance concentrate themselves upon the interface of contact between the two phases. This accumulation or surface concentration of a substance at the boundary between two heterogeneous phases is termed *adsorption*. Adsorption is, therefore, merely a physical concentration of atoms or molecules on other molecules or surfaces, and any substance, dissolved in a fluid which is in surface contact with another phase, will be concentrated or adsorbed on that surface or series of

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surfaces if it can lower surface tension and thereby decrease the free energy. If there are several dissolved substances, all will be adsorbed at the interface, but in proportion to their power of lowering surface tension; and, in consequence, one adsorbed substance can be displaced by another which has greater powers of lowering surface tension, and, if a dissolved substance such as certain inorganic salts raises surface tension, its concentration at the interface will actually be lowered—negative adsorption. Adsorption requires time for its accomplishment. The rate of adsorption increases, but the quantity of adsorption diminishes, with rise of temperature. Being merely a concentration, adsorption is usually reversible, but it may become irreversible, as was shown by Ramsden's experiments in which white of egg, saponin, and quinine were deposited in solid form in contact with air to form a rigid surface film.

Hitherto we have dealt purely with surface energy as the cause of adsorption, but any process which diminishes free energy—whatever its nature—at an interface will tend to take place. The boundary face between two immiscible phases is always the seat of electrical forces, and, as we have seen, colloidal particles possess electrical charges; hence, any process which can reduce the electrical energy at the surface will be liable to occur, and, if the surface has a negative charge, for example, positively charged bodies (cations) will be adsorbed upon it so as to annul the charge. Similarly, chemical reactions will be favoured at interfaces if they lower the chemical potential there. The various forms of energy which give rise to adsorption may of course all be present at the same surface. It will now be apparent that we may obtain adsorption compounds consisting of, for instance, two colloidal particles of opposite electrical charge in close apposition or agglutinated. Such compounds differ from true chemical combinations in the variability of their composition. Artificial laccase, an oxidizing enzyme, is an example of an adsorption compound between gum and colloidal manganese hydroxide. Adsorption takes place without any chemical action, but a chemical reaction may follow adsorption and be directly attributable to the close approximation of the reacting molecules brought about by the adsorption; thus, in catalytic reactions, two substances capable of reacting are adsorbed on the surface of a third, where, being in intimate contact, they combine without altering the adsorbing

surface. The rate of such chemical reactions is naturally dependent on the rate of adsorption.

Preparation of colloids.—The essential object to be attained in preparing a substance in the colloidal state is a definite degree of dispersion; the use of freshly distilled water and absolutely clean hard glass apparatus is essential. Only a few methods can be given here: (1) By adding a solution to a liquid in which the solute is insoluble, e.g. an alcoholic solution of gamboge added to water. (2) By the action of various reducing agents—as hydrogen, carbon monoxide, formaldehyde, hydrazin hydrate—on solutions of the salts of heavy metals, e.g. a 1-per-cent. solution of gold chloride is neutralized with dilute sodium carbonate and a weak (1-in-4,000) hydrazin hydrate solution added drop by drop. By this method the suspensoid must be prepared in dilute solution and, before concentration, the electrolytes dialysed out. (3) Many other chemical methods are used, e.g. hydrolysis, action of sulphuretted hydrogen, tannic acid, etc. (4) Electric-arc dispersion. Two electrodes, e.g. of gold wire, are adjusted below water to a 1-mm. arc and a current of 30–40 volts and 8–10 amperes passed; while the arc lasts purple red clouds are evolved from the cathode wire. Svedberg employs the induced current from a large faradic coil connected in parallel with a glass condenser, the electrodes dip into the vessel containing the fluid, and the metal is placed in small grains at the bottom of the vessel. (5) Mechanical disintegration has been used by Kusel, who ground metals finely between ball mills and treated them alternately and repeatedly with strong alkalis and acids. For details of the preparation of individual metallic sols the reader is referred to Martindale and Westcott's "Extra Pharmacopœia," 1920, i, 349.

The precipitating action of electrolytes can be delayed and suspensoids stabilized by adding an emulsoid (protective colloid, e.g. peptone, gelatin) to the suspensoid; the effect is due to adsorption of the emulsoid on the suspensoid particles; by this means dry suspensoids can be obtained.

The majority of emulsoids are natural organic products and either exist as such in nature or, if solid substances, they form emulsoid systems in contact with a suitable dispersing medium. Silicic acid is an inorganic emulsoid. The following substances, when immersed in water, form emulsoid sols, each having characteristic properties, viz.:

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starch, dextrin, gums, agar-agar, Irish moss, albumins, globulins, caseinogen, gelatin, many dye-stuffs, etc.

Colloids in relation to physiology, pathology, and pharmacology.—The following is merely a brief synopsis of some of the biological reactions which are probably based on the physical properties of colloidal matter. Fuller information is obtainable in Bayliss's "Principles of General Physiology," Beckhold's "Colloids in Biology and Medicine," and the First, Second and Third Reports of the British Association on "Colloid Chemistry."

Most of the body tissues are composed of colloidal systems, and even a unicellular organism, e.g. a leucocyte or amoeba, exhibits colloidal characteristics. Its protoplasm in the ultramicroscope shows minute particles in Brownian movement. In contact with fluids it forms an adsorption surface membrane and moves by putting forth pseudopodia when in contact with a solid substance, phenomena derived from surface tension, which also gives rise to internal pressure in the organism; thus, the protrusion of a pseudopodium is due to lowering of surface tension at some local point on which the internal pressure then acts. Protoplasm is therefore a sol, but it may assume the gel condition in spore formation or on death, just as a gelatin hydrosol may become a hydrogel. Food, e.g. a bacterium, is first encountered and then engulfed by a process of softening and rupture of the surface membrane; this is again a surface action, and preliminary adsorption may augment it, as will also agglutination of the food particles. As Ledingham has shown, the postulation of opsonins to explain phagocytosis is unnecessary. After ingestion, the food is surrounded by a vacuole into which enzymes are secreted; at first, these are acid and serve to kill the bacterium; later, they are alkaline and digestive—undigested material is extruded. Useful products of digestion are retained and useless products excreted by the surface membrane, which acts, like an artificial semi-permeable membrane, as a sieve. Since the surface membrane is formed by adsorption, it will contain the cell constituents in proportion to their powers of lowering surface tension, and it therefore consists largely of lipoids and similar fatty substances which are powerful agents in reducing surface tension, but its composition will also vary with the composition of the external fluid; its permeability will therefore be changeable. The cell membrane is always readily permeable

by lipid soluble substances, but in specific conditions it is permeable by substances not belonging to this category. Colloids have probably been the important factor in determining the percentage of salts in the tissues of vertebrates, because the percentage of sodium chloride varies little in the wide range of species between fish and man, e.g. 0.7–0.9 per cent. This strength represents the maximal amount which the colloids of the cells and plasma are capable of adsorbing; any excess remains free in the plasma till excreted. Certain cells have specific adsorptive powers, e.g. the bone-forming tissues adsorb calcium and phosphate ions.

Enzymes as colloids.—In the living organism many catalytic actions are due to enzymes. These do not as a rule originate chemical reactions; they probably only hasten reactions which would proceed slowly without their help. Enzymes exist in colloidal form; their action in a solution containing two substances capable of interaction takes place, first, by the fusion of the molecules of the substance with the particles of the enzyme; secondly, the molecules are adsorbed by the enzyme particles; and thirdly, chemical action takes place between the adsorbed molecules; the enzyme itself plays merely the part of an adsorbing surface. The rate of the reaction follows the parabolic curve of adsorption, i.e. the velocity increases to a maximum at which it remains constant—the limit of which the adsorbing surface is capable has then been reached. With low temperatures slow diffusion delays enzyme action; but, as adsorption is generally a more rapid process than chemical action, it is the latter factor which ultimately determines the speed of enzyme actions. The inactivation of enzymes during their action may be due to their precipitation by electrolytes formed during the chemical actions they are promoting, or to the presence of alcohol, saponins, etc., which lower surface tension greatly and are therefore adsorbed on the enzyme to the exclusion of the reacting molecules; they are also sensitive to changes in the H-ion concentration: thus, trypsin may be inactivated by producing amino-acids which decrease the hydroxyl concentration favourable to trypsin. Enzymes can accelerate both hydrolytic and synthetic reactions, and in the case of reversible reactions the position of equilibrium is usually dependent on the concentration of the products in water; thus the amylase of the liver cells when glucose is in small percentage in the blood will hydrolyse

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the glycogen store of the cell, but, when the amount of the glucose in the blood is high, synthesis of glycogen will take place.

Peroxi-dases are oxidizing enzymes which from peroxides liberate active oxygen. Peroxi-dases exist in plants and in the body tissues; in plants a peroxide is also present, but it is formed only by autoxidation of another spontaneously oxidizing substance—an oxygenase—in the presence of oxygen. The active oxygen liberated may be demonstrated by its effect on tincture of guaiacum or other substrates. It has been found that colloidal hydroxides of iron, manganese, and copper act like peroxidases, and it is probable that peroxidases are very active compounds of one or other of these metals.

The rôle of colloids in the blood.—The phenomenon of bloodclotting is related to the colloidal condition of the blood constituents; actually a precipitation and coagulation of fibrinogen takes place under the influence of electrolytes, among which the calcium salts are the main factor.

Between the blood-vessels and the tissues, interchange of crystalloids, but not of colloids, can take place; thus the blood proteins are not utilized as food by the tissues. The blood colloids maintain the osmotic tension of the blood, and so permit the interchange of water and crystalloids between the blood and the tissues. As the osmotic pressure of the blood colloids is 40 mm. Hg as compared with the arterial blood-pressure of 120 mm., it follows that the osmotic pressure of the colloids cannot draw fluid into the blood until the arterial pressure is less than the osmotic pressure of the colloids; this exists first in the capillaries. Before this stage is reached, filtration of water and crystalloids outwards into the tissues will occur. A similar sequence of events takes place in the glomerulus of the kidney, where filtration of the crystalloids with water takes place so long as the blood-pressure is above 40 mm. Hg; below this level, urinary secretion ceases, and to restore it we must either raise the blood-pressure or decrease the osmotic tension of the colloids; the latter result may be attained by the injection of isotonic saline to dilute the blood. If, along with the saline, we introduced a colloid in the same osmotic tension as the blood colloids, secretion of urine would not recommence, since the colloid would annul the effect of the saline. The normal plasma salts, together with the glucose, amino-acids, etc., which are filtered from the blood

in this way are reabsorbed from the kidney tubules, but excretory products, e.g. urea, are not reabsorbed.

The conveyance of oxygen and carbon dioxide by the blood is not yet fully elucidated, although it is the hæmoglobin which is the essential medium. According to one theory, chemical combination takes place between oxygen and hæmoglobin with formation of a homologous series of compounds. It has been suggested also that the iron in hæmoglobin may play the part of a peroxidase and that lecithin may form the autoxidizable peroxide. On the other hand, adsorption by the colloid hæmoglobin has been put forward to explain the conditions; the dissociation curve of oxyhæmoglobin in water is a hyperbola, but in the presence of electrolytes the curve approaches the adsorption parabola, and the ease with which the oxygen of hæmoglobin is displaced by the more readily adsorbed gases, carbon dioxide, carbon monoxide, etc., points rather to an adsorption phenomenon; the effect of temperature, namely, that at higher temperatures oxygen is taken up in smaller quantity but with greater rapidity, coincides with the adsorption theory.

Toxins, antitoxins, and vaccines as colloids.—In the toxin-antitoxin reaction the reacting substances are colloids, and when examined by the ultramicroscope, the reaction results in precipitation. If antitoxin be added to diphtheria toxin the amount neutralized depends on whether it is added in large quantities suddenly, when the amount neutralized is large, or slowly, when a smaller amount is neutralized. Similar "tolerance" effects are known to occur with colloids, e.g. in precipitation of suspensoid sols by electrolytes and in the "salting out" of albumin. The combination of toxin and antitoxin has been found to follow the laws of the adsorption formula, but the specificity of these reactions remains so far unexplained on a colloidal basis.

Emulsions of bacteria are probably suspensoids protected by an emulsoid sol. They are not very sensitive to electrolytes, but these do precipitate them. The addition of the immune serum renders a bacterial sol more sensitive to electrolytes, hence the immune serum appears to displace the protecting emulsoid and permit the bacterial particles to agglutinate. The effect seems to result from adsorption. The phenomena of the Wassermann reaction and of other hæmolytic phenomena can also be explained on the adsorption basis.

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Anaphylaxis is the extreme sensitiveness developed by an animal to a foreign protein eight to twelve days after it has been injected with the protein. Exhibited most commonly after the injection of antitoxins, it occurs also with apparently innocuous foreign proteins such as egg albumen and horse serum. As a rule anaphylaxis is specific, but it is not necessarily so. The action appears to consist in a colloidal precipitation process at the surface of the sensitive cells, by which their semi-permeability is more or less destroyed. The anaphylactic substance is adsorbed on the tissue cells, so that even the excised uterus of the animal is found to be sensitive to the original protein and specific to it although only in a quantitative way. If a uterus sensitive to horse serum has given a powerful contraction in response to a fairly large dose, it will then be found incapable of response to a second dose—a phenomenon known as “desensitization.” What has happened is that the antigen has become adsorbed on the sensitive tissue so as to exclude the action of further doses. The contraction resulting from the interaction of a sensitized muscle-fibre with the antigen is a consequence of the increased permeability of the cell membrane, and calcium salts, which lessen the permeability of membranes, decrease the reaction.

Lange's colloidal gold reaction.—This reaction is performed with cerebro-spinal fluid which is diluted with 0.4-per-cent. saline solution, starting with 1-in-10 dilution and increasing by geometrical progression to 1-in-5,120. The colloidal gold solution is then added. No change occurs with normal cerebro-spinal fluid, but with abnormal fluids the red colour (0) may be changed to (1) red-blue, (2) purple, (3) blue, (4) pale-blue, or (5) colourless. In general paralysis and multiple sclerosis the first four dilutions are decolorized, giving a numerical colour series of 5555421000 (paretic type); in tabes and cerebro-spinal syphilis the series is 3334310000 (luetie type). The reaction is not pathognomonic of cerebro-spinal syphilis, and is useful mainly to differentiate dementia paralytica.

Colloids in relation to water supply.—In a contaminated water supply organic impurities exist in colloidal form, and the adsorptive power of sand and charcoal in large filter-beds is the means by which they are removed. Application of the methods of precipitation is also made, e.g., by the addition of alum or ferrous salts, etc.

Colloids in relation to drug actions, etc.—From the mode of action of certain drugs it is probable that adsorption determines to some extent their affinity for certain cells, and modification of the surface tension and permeability of the cell may play a part in determining their action. Strophanthin and veratrine appear to act while temporarily adsorbed on the heart tissue. In the case of anæsthetics, their first action is to decrease the permeability of cells, and this leads to a state of inhibition such as is produced by deprivation of calcium salts for the same reason; later, however, anæsthetics cause an increase of permeability which is irreversible and leads to death. Macdonald suggests that the phenomena of excitation and inhibition exhibited by a neurone may be due to release from adsorption of charged electrolytes (K salts) which thus confer a positive charge on the colloids and a negative charge on the adjacent section of nerve, and there is evidence that muscular contraction also originates from alteration in the surface tension between the fibrillæ and the sarcoplasm of the muscle cell. It may be that lactic acid alters the electrical charges at the boundary surface, where an alteration of surface tension will then occur, leading to shortening of the muscle-fibre.

Colloids in therapeutics.—The application of colloids to the treatment of disease is of comparatively recent date, and so many different substances in colloidal form have been employed in such a large variety of ways that it is difficult to form reliable conclusions as to their specific advantages over drugs exhibited in other forms. Chemically prepared colloids appear to be equally efficacious with those prepared by electrical dissociation. The presence of a protecting colloid must necessarily have an influence on the activity of the sol, but no reliable statements are available indicating how these protecting colloids modify the action of the internal phase. As the protecting colloid is adsorbed on the suspended particles, it must necessarily decrease the adsorption of other bodies on the particles unless these bodies are capable of lowering the surface tension more, and thus of displacing the adsorbed protective colloid. It is not usual for manufacturers of colloidal remedies to state which protective colloids have been employed, but gelatin or peptone is commonly used. In order to render colloidal metallic solutions isotonic, sodium chloride or sugar is added to them; in many cases the electrolyte causes

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instability of the colloid; if so, it should be added immediately before use.

One must guard against assigning to colloids powers which they cannot possess; thus, the statement that no drug can produce its full therapeutic effect until it is in colloidal form, cannot be accepted as a generalization, even although the tissue reactions are of a colloidal nature. Our knowledge of colloids and their reactions in the body is incomplete, but therapeutic evidence indicates that substances in the colloidal state possess certain advantages unobtainable from molecular solutions.

General action of colloids.—Colloids have been administered internally—by mouth, hypodermically, intramuscularly, and intravenously. Used by the first three methods the administration gives rise to no important effects beyond the pain which may occur from hypodermic or intramuscular injection. Practically all colloids intravenously injected cause a reaction commencing with a rigor lasting twenty to thirty minutes. At this stage the blood-pressure rises 10 to 20 mm. and the temperature increases to 100° or 104° F. in two to four hours; pulse and respiration are rapid; sweating occurs, and often headache and sickness. At first there is a diminution in the number of leucocytes, but during the rise in temperature leucocytosis is observed, affecting both the mononuclear and polynuclear cells, and persisting for forty-eight hours. This leucocytosis may be the basis of the antiseptic action of the colloidal metals in the body. The reaction subsides rapidly, and varies in degree with different colloids. It has been stated that adrenalin injections mitigate the reaction.

In individual diseases the dynamics of the effect of colloids is obscure. There is considerable similarity in their effects on disease, and it has been suggested that the metallic phase of the sol is of less importance than the protecting colloid. Auld (*B.M.J.*, 1918, i, 195) has shown that injection of peptone can produce therapeutic effects similar to those exhibited by colloidal platinum, and suggests that the effect of colloidal metals in infective diseases may be due to pyrogenic action in which the protecting colloid plays the part of an antigen and liberates the pyrogenic substance, anaphylotoxin, from the plasma. McDonagh (*B.M.J.*, 1917, i, 648), on the other hand, regards the body proteins as protective agents in virtue of a surface action, associated with oxidation and reduction processes in which iron plays a part, and suggests that the metallic colloids act as agents promot-

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ing oxidative processes while the non-metallic colloids—iodine and sulphur—promote reduction. On this theory the action of metallic sols may be considered as in the nature of a peroxidase reaction.

Colloids in surgical shock.—Normal saline solution causes merely an evanescent increase in the blood-pressure and blood-volume when these are reduced as the result of hæmorrhage, since the lowered osmotic tension causes a greater secretion of water from the kidneys. In order to maintain the blood-volume, Bayliss (*Proc. Royal Soc.*, B, 1911, lxxxiv, 229) advised the use of an isotonic 0.9-per-cent. saline containing a colloid in the proportion which would give the fluid the osmotic tension of blood, e.g. gum acacia 6 per cent. About 500 c.c. of such gum infusions are injected intravenously in fifteen minutes and repeated if necessary. Gum infusions maintain the blood-pressure and blood-volume for several hours and were used with great benefit during the War. There is little positive evidence that blood transfusion is more advantageous.

Metallic and non-metallic colloids.—These have been used most largely in the treatment of various infective disorders, and include the colloidal preparations of silver, gold, platinum, arsenic, mercury, iron, manganese, palladium, iodine, sulphur, copper, selenium, and antimony. The indications for their use are generally wider than those of the corresponding non-colloidal preparations; published reports demonstrate that these preparations are relatively less toxic in their action than the corresponding non-colloidal preparations, and suggest that in the future they will have a wider field of usefulness. W. J. DILLING.

COLOBOMA, CONGENITAL (see EYE, CONGENITAL ANOMALIES OF).

COLON, CANCER OF (see INTESTINE, NEW GROWTHS OF).

COLON, DILATATION OF.—Of this condition four main varieties are met with:

1. Acute non-obstructive dilatation.
2. Distension by impacted fæces.
3. Obstructive dilatation.
4. Idiopathic dilatation.

1. ACUTE NON-OBSTRUCTIVE DILATATION.—This variety is frequently met with as a complication of acute infective fevers, and it also occurs as a sequel to abdominal operations. It is due to gaseous distension, the contents

gas consisting chiefly of carbon dioxide. In many cases the distension is extreme, the bowel-wall when exposed either at operation or post-mortem being found almost transparent and extremely thin. Occasionally even the peritoneal coat may have ruptured. In the post-operative form it is possible that some degree of peritonitis is always present, and is responsible for the paralysis of the bowel-wall, whilst in the meteorism of fevers the general toxæmia leads to similar paralysis. When distension has begun it is liable to increase owing to angulation and pressure of the distended parts against one another. **Diagnosis** is easy. The abdomen is distended and tense; it is tympanitic on percussion, and there may be displacement upwards of the liver and the spleen, and also of the thoracic viscera.

Treatment will vary somewhat with the cause; for example, meteorism in enteric fever is sometimes alleviated by a change of diet. Carminatives by the mouth are seldom of use, but may be tried. Of more value are enemata containing turpentine, or 2 dr. of liquor ammoniæ in a pint of water. The latter must be immediately siphoned out again. Pituitrin hypodermically (1 c.c.) is often useful, as are hot stupes applied to the abdominal wall. A rectal tube seldom does good. As a last resort surgical treatment may be required.

2. **DISTENSION BY IMPACTED FÆCES.**—In this condition enormous accumulations may result and simulate tumours.

3. **OBSTRUCTIVE DILATATION.**—This may occur as an acute or chronic condition. Chronic dilatation is met with in some cases of extreme ptosis of the bowel, in stricture, in chronic volvulus, and as the result of adhesions. The treatment in each case is for the primary condition.

4. **IDIOPATHIC DILATATION OF THE COLON** (*Syn.* Hirschsprung's Disease; Congenital Dilatation of the Colon).—This rare disease is most frequently met with in childhood, 40 per cent. of the cases being under 10 years of age. It is, however, also encountered in adults.

Etiology.—The disease is undoubtedly congenital, but its cause is unknown. In a few cases an actual obstruction has been found in the lower bowel, such as stricture of the rectum. In nearly 80 per cent., however, no obvious obstruction is present. Some attribute the dilatation in these cases to a neuro-muscular defect leading either to spasm or to defective

peristalsis of the lower bowel. Another and more probable view is that the mesentery of the pelvic colon is too long, and allows kinking of the bowel to take place, and consequent gradually increasing obstruction.

Pathology.—The dilatation is often enormous, and may involve the whole colon, but it is usually local, the pelvic colon being most often involved. The distended part may fill almost the entire abdomen: its muscular wall is always much hypertrophied; and its diameter may reach 18 cm. or even more.

Symptomatology.—The symptoms include (1) *Abdominal distension*; loops of the colon disposed vertically may be seen through the thinned abdominal wall, and peristaltic movements may be present. (2) *Constipation* dating from birth, with occasional intervals of diarrhoea; the constipation is unaffected by aperients. (3) *Abdominal pain* is often complained of, and is usually capable of relief by washing out the bowel. (4) *General maldevelopment*, in severe cases with progressive emaciation and loss of appetite.

Diagnosis.—If any question exists as to the real nature of the condition, the injection of a bismuth emulsion into the colon, followed by radiographic examination, will usually remove all doubt.

Treatment.—In severe cases surgical treatment offers the only prospect of cure. Excision of the distended portion, though a serious operation, gives the best results. Colotomy is very dangerous and should not be performed, and it is doubtful if a short-circuiting operation would give more than temporary relief. Some recommend emptying the bowel by squeezing out its contents through the anus, and state that subsequent lavage, with aperients, steadily persisted in, gives relief. In milder cases the injection of olive oil at night, followed by colonic lavage in the morning, and accompanied by massage, is to be recommended. A suitable abdominal belt may also aid by keeping the bowel in position. Purgatives are usually of no value, but, if tried, a pill containing strychnine, belladonna, and aloes is the most suitable.

T. G. MOORHEAD.

COLOUR VISION.—Variations in sensation are aroused when lights of different wavelengths fall on the retina. The combination of light from all parts of the spectrum gives rise to the sensation of white, but the sensation of white may also be aroused by stimulation by three of the components of the spectrum

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alone, while by modifying the mixture of these components, either by diminishing or omitting one or other of them, any variation in colour sensation can be produced. The three colours whose presence is necessary to produce the sensation of white are red, green, and violet, and by suitable admixture of these all other colour sensations can be produced in a person possessing normal colour vision. Of all the problems in which physics, physiology, and psychology are concerned, none is more controversial than the relationship between the sensation of colour and the physical ethereal vibrations which fall on the retina. In our present state of knowledge it is impossible to be dogmatic. We cannot make positive assertions as to what part is played by physiological processes in the retina, or by differentiated elements in the retinal sensory epithelium, or in the primary or secondary visual neurones in the brain. What we do know definitely is that at one end there is a physical process, light falling on the retina, and at the other end a psychical process which we call a sensation of colour. We know, too, that differences in the condition of the eye will cause variations in the sensibility to colour; that when the eye is adapted for darkness the sensations produced differ from those excited when the eye is light-adapted. Anything which lowers the functional capacity of the retina lowers its power of colour discrimination. There exists a very considerable proportion of the population (mainly males) who are deficient in the discrimination of colours.

The loss of the power of colour discrimination (**acquired colour blindness**) may be the result of disease or degeneration of the retina, or of the conducting fibres of the optic nerves, or of their cerebral connexions. In tobacco blindness, for example, a central scotoma for colour is the most important diagnostic feature; in retrobulbar neuritis some central or paracentral colour loss often persists when all other visual functions seem to have recovered completely; in pituitary growths a bitemporal colour hemianopia may precede the development of an ordinary hemianopia.

It is, however, when we come to deal with **congenital defects of colour vision** that we find ourselves in the field of greatest controversy. It is beyond the scope of this article to attempt an exposition of the various theories of colour vision, but it is essential for every medical man to realize that almost 4 per cent. of the male population are colour blind, and

that full colour vision is essential in certain callings which give employment to large numbers of men, the two most important being the railway and marine services. The two colours which it is most necessary that men in these services should be able to discriminate are red and green, and it is these which the colour-blind are most likely to confuse.

Tests of colour vision.—From the practical point of view the important thing is to determine whether a man possesses the power to discriminate properly between red and green in all the circumstances necessary for his calling. For many years the ordinary method of testing was by means of different-coloured wools, and even to-day this method, if carefully used, is of great value. There is no doubt, however, that dangerously colour-blind men have occasionally succeeded in passing the test, an increased power of discriminating differences of saturation or luminosity probably compensating to a certain extent their loss of power to discriminate hues. In place of wools, Stilling used pseudo-isochromatic diagrams in which patterns of one colour were arranged on a background of its confusion colour. Several different tests based on this principle of Stilling's are now to be obtained, the most recent being Edridge Green's cards, in which letters composed of stars and blotches of irregular shape in one colour are placed among similar stars and blotches of the confusion colours. If colour vision is defective the patient is unable to see the letters.

Any test which depends on naming the colour is to a certain extent unsatisfactory, as inability to name a colour may arise from ignorance rather than from colour blindness. Yet from the practical standpoint a lantern test is essential. It approximates most closely to what is required of railway men or sailors in their employment, and it should be always used as part of the test in such cases. The lantern most readily available in this country is Edridge Green's, and it should be used as a final test in the conditions of partial dark adaptation in which it is most likely that the men will be called upon to discriminate between signal lights.

It should be added that as one of the common causes of colour blindness is heavy smoking, and as with advancing years the power of colour discrimination diminishes, a periodical re-examination of the colour sense in all men whose calling demands good colour vision is necessary.

LESLIE PATON.

COMA.—A patient is said to be in a state of coma when stimuli fail to arouse him to consciousness, when liquids placed in the mouth are not swallowed, and the conjunctival reflex is absent. It is obvious that practically every morbid condition that may end fatally can give rise to coma if its development be such that death or loss of function of the cerebrum precedes that of the organs of circulation and respiration and their nervous mechanisms.

From the practical point of view the consideration of coma may be limited to those cases in which the patient is seen for the first time in an unconscious condition and it is necessary to arrive at a diagnosis of its origin. The **examination** of such cases should be conducted with thoroughness and attention to systematic procedure. The history must be minutely inquired into, the possible medico-legal aspect of the case being kept in view as well as the purely diagnostic problem. The general aspect of the patient is noted, the head and body are examined for injury, and attention is paid to the presence of any stain on the hands and mouth, or traces of vomit on the clothes. The breath must be smelt carefully, its odour being often the only key to the diagnosis. Next examine the pupils—their size, equality, and reaction to light. Test the conjunctival and corneal reflexes. Note any deviation of the eyes. Examine next the heart and lungs. The type of breathing should be observed, together with the symmetry of the respiratory thoracic movements and the effect of respiration on the facial muscles; flaccid paralysis of one side of the face causes sucking-in of the *alæ nasi* during inspiration and blowing-out of the cheek during expiration. Spasm of the neck muscles is looked for by testing the rigidity of the head. The skeletal muscles are palpated and the limbs moved to detect the presence of spasticity or flaccidity. The deep reflexes and the abdominal and the plantar reflexes are compared on the two sides. The temperature is taken in each axilla. Finally, the fundus oculi is examined and a specimen of urine is drawn off for examination. Should the preliminary examination indicate its necessity, a lumbar puncture should be performed.

The **causation** of coma may now be briefly considered. It may be due to poisoning of the cerebrum by an endogenous or an exogenous toxin, or to the direct effect of a gross nervous lesion. Generally speaking, the effects of poisoning involve both cerebral hemispheres

equally and there is no difference in the state and reactions of the two sides of the body—the plantar reflexes are of the normal flexor type. The most common *exogenous toxins*—alcohol, carbolic acid, prussic acid, chloroform, ether, opium, paregoric, and turpentine—can usually be recognized by their smell. The value to be assigned to the smell of alcohol will naturally depend upon whether it has been given as a restorative by the patient's friends. The pupils in opium poisoning are pin-point and do not react to light. Belladonna and its allies produce equally dilated inactive pupils. The pupil in alcoholic poisoning is dilated but reacts to painful stimuli. Aconite and digitalis poisoning also cause mydriasis; in the former case, however, the slow onset of coma will generally allow of some history being obtained from witnesses. In it the patient complains first of tingling, and the pulse is very minute. Poisoning by digitalis is unlikely to occur except in a patient who has been taking it for cardiac disorder; signs of diarrhoea and vomiting will generally be found; the pulse may be very slow and full, but is more often rapid and irregular. The possibility of ptomaine poisoning must be borne in mind. Evidence of diarrhoea and vomiting is here of importance.

Diabetic coma is characterized by the smell of acetone in the breath and the emaciation of the patient, but it must be remembered that in fat subjects a very short period of starvation will cause abundant acetone excretion; the presence of sugar in the urine will clinch the diagnosis. In *uræmic* coma the breath has a characteristic fishy smell due to trimethylamine, the arteries are often thickened, and there may be retinal hæmorrhages. Occasionally the uræmic poison causes cerebral symptoms resembling those due to a gross nervous lesion, and unilateral or bilateral extensor plantar responses, flaccidity or rigidity, and the occurrence of epileptic fits may lead to a wrong diagnosis.

The most common form of gross nervous lesion is *cerebral hæmorrhage*. The onset is sudden, the breathing stertorous, and the blood-pressure high. A more marked flaccidity on one side with a unilateral extensor plantar response points to a unilateral lesion. In such a case the head and eyes are generally turned away from the hemiplegic side, unless there be spasm of the limbs due to cortical irritation; then the head and eyes are turned to the affected side. The pupils are generally dilated

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and unequal, the larger pupil being on the same side as the hæmorrhage. In later stages, when the cerebral hæmorrhage is exerting pressure on the mid-brain, and in cases of primary mesencephalic hæmorrhage, the pupils are pin-point.

Thrombosis of the cerebral arteries produces a condition of coma that is practically indistinguishable from that due to hæmorrhage. The seizures of general paralytics are, again, often impossible to distinguish from those due to cerebral hæmorrhage or thrombosis.

Epileptic fits are frequently followed by a comatose condition; when the fit has involved one side of the body more than the other there may be all the signs of hemiplegia, and the condition cannot be differentiated from a gross cerebral lesion.

Coma due to *intracranial pressure* from neoplasms, abscesses, or gummata will generally be accompanied by optic neuritis.

The coma of *meningitis* is distinguished by rigidity of the neck muscles and retraction of the head. Kernig's sign may be sometimes elicited, and the performance of lumbar puncture will generally make the diagnosis clear. The coma of lethargic encephalitis is seldom encountered without a definite history of the prodromal symptoms.

Enough has been said to make it clear that the prognosis to be given must be very guarded.

The treatment of coma is dealt with under the various diseases and intoxications that may cause the condition. There is, however, one golden rule—that is, never to leave a case of coma without attendance.

F. L. GOLLA.

COMBINED DEGENERATION, SUB-ACUTE.—A disease characterized by the occurrence of diffuse degeneration and sclerosis in the white matter of the spinal cord. The degenerative changes begin in the mid-dorsal region, and here the sclerosis usually attains its maximum transverse extent, involving the dorsal, lateral, and anterior columns; at other levels the process is much less severe, and may be confined to parts of the dorsal and lateral columns.

The early lesions in the mid-dorsal region consist of separate foci of degeneration, which gradually increase in size and coalesce with one another to produce an extensive involvement of white matter at this level. In other parts of the cord the changes are due partly to the addition of new foci, but chiefly to an ascend-

ing and descending degeneration in the various afferent and efferent fibres whose axis-cylinders are destroyed in the foci of the dorsal region.

Etiology.—The cause of the condition is obscure. It is usually accompanied by anæmia, frequently of a very severe grade, and it was at one time thought that the spinal-cord changes were secondary to this. It is now held that both the cord changes and the anæmia are more likely due to a third factor, probably toxic in character, the exact nature of which is not yet determined.

Symptomatology.—The disease is usually gradual in onset. It attacks both men and women, and occurs in the mid-period of life. Early in its course the patient complains of a constant tingling or numbness in his toes or feet, and as the disease advances this tends to spread from the distal parts of the limbs towards the trunk. Sometimes other subjective sensations are present, as pains of an aching or shooting character, girdle sensations about the trunk, or feelings of constriction in the limbs.

Sooner or later impairment of the various forms of sensibility occurs, and especially of the sense of position and the recognition of passive movement. The sensory loss begins in the distal parts of the limbs and extends thence to more and more proximal parts, the lower limbs usually being involved earlier and to a greater extent than the upper, while on the trunk it will be found that the cutaneous loss spreads gradually from lower to higher root levels.

Early in the disease the patient complains of weakness or stiffness in his legs, or unsteadiness in walking, and gradually a condition of ataxic paraplegia develops. In some cases he may complain early of weakness or clumsiness in his hands.

The ataxic paraplegia results from a combination of the symptoms due to the lateral and dorsal column degenerations, and varies considerably in its character according to the relative involvement of these two columns. It may be, therefore, spastic or flaccid in type. Frequently there is a spasticity in the earlier stages which gives way to a flaccidity later in the disease; sometimes the change from the one to the other may be very abrupt. In the spastic stages the knee-jerks and ankle-jerks are brisk, ankle-clonus may be present, and the plantar responses are of the extensor type. As the flaccidity supervenes, the deep reflexes become diminished or absent, while the plantar

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responses remain extensor. The paraplegic symptoms are usually progressive, and lead finally to a condition of bedridden paralysis.

Sphincter troubles are important and early symptoms in the disease. There is usually, first, a difficulty in the control of the bladder, a precipitancy or incontinence of urine; later, control of the rectal sphincter may be impaired or lost also.

Other symptoms are due to the anæmia that is frequently present. It is common to find that the patient is pale, even of the lemon-yellow colour associated with pernicious anæmia, that he is short of breath on exertion, has headaches or attacks of giddiness, and suffers from indigestion or attacks of vomiting. In the late stages the anæmia may become profound, mental symptoms develop, and the patient become delirious; his extremities are then cedematous, his heart fails, and the delirium sinks into coma and death. In other cases the anæmia may be less severe and the course of the disease more prolonged.

Prognosis.—The disease is steadily progressive. Acute and chronic cases occur, however, and the average duration in these two classes has been estimated at six months and six years respectively. The presence of much anæmia hastens the end.

Diagnosis.—In the differential diagnosis a number of conditions have to be considered. The numbness and occasional lightning pains, the sensory loss, ataxia, inco-ordination, and sphincter trouble may suggest tabes dorsalis; the paræsthesia and signs of spastic paraplegia may point to disseminated sclerosis; and the distal distribution of the sensory loss may suggest polyneuritis. The intramedullary and extramedullary cord lesions that give rise to paraplegia, such as myelitis, compression lesions, spinal tumours, may have to be considered, and of these, malignant disease with diffuse involvement of the cord or meninges will need the most careful differentiation. The degenerative conditions affecting the dorsal and lateral columns of the cord—for example, Friedreich's disease—may enter into the differential diagnosis also; and lastly, in those cases of sub-acute combined degeneration which come under observation early, and in which the progress of the disease is slow, and definite organic signs are late in appearing, various functional conditions may have to be excluded.

There is no treatment of any known value in arresting the morbid process in the cord. The association of the disease with anæmia

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and the frequent association of that condition with chronic septic infections suggest the utility of vaccine treatment, but efforts in that direction have not yet met with any noteworthy success. Of all the drugs that have been used, arsenic is the one on which most reliance has been placed, probably because of its value in combating the anæmia. Apart from radical treatment, however, much may be done for the patient by rest, food, and tonic treatment, and by various measures directed to the alleviation of his symptoms and the prevention of complications.

P. W. SAUNDERS.

COMPRESSION PARAPLEGIA (*see* SPINAL CORD, LOCAL LESIONS OF).

CONCOMITANT STRABISMUS (*see* STRABISMUS).

CONCUSSION AND COMPRESSION (*see* HEAD INJURIES).

CONFABULATION.—A distortion of memory whereby events are recollected which actually never took place. It is generally accompanied by an amnesia, the gap in the memory being filled by the fictitious reminiscences. Confabulation occurs notably in certain alcoholic and senile psychoses, and is a variety of the disturbance of memory known as paramnesia. (*See* MEMORY, DISTURBANCES OF.)

BERNARD HART.

CONFUSIONAL INSANITY (*syn.* Insanities of Toxic and Infectious Origin; Insanity of Exhaustion; Traumatic Insanity).—Confusion may be defined as consisting in diminution of mental clarity and awareness in various degrees, so that orientation in time and space may be ill-defined or even lost, and the sense of personality diminished or perverted. In such a degree may this diminution or loss obtain that there may be complete mental palsy or stupor. On the other hand, though the highest controlling mental levels are inhibited, there may be unbridled activity of those that are lower and more automatic, so that there ensues considerable excitement and agitation, often accompanied by hallucinations affecting various senses.

Etiology.—While it is likely enough that heredity has provided the patient with a mental make-up more than usually prone to take a morbid turn upon provocation which would not affect a normal individual, it cannot be held to have so important a place in etiology as in certain other psychoses. The abnormal

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and trying conditions to which a patient has recently been exposed are of greater importance, and their special investigation is of interest as concerning prophylaxis. The two outstanding causes are intoxication and exhaustion, and it is not improbable that the second may in truth be included under the first. Mental confusion is associated with acute infective illnesses, and with morbid states in which intoxication, presumably by smaller and less virulent doses, goes on over a considerable period of time, as in certain cases of tuberculosis, syphilis, or renal or gastro-intestinal inadequacy. It may accompany or succeed every sort of stress which can exhaust the human frame: overwork, whether physical or mental; energy devoted to pleasure in excess, as in a "Society woman" during the course of a London season; privation and starvation; emotional states of a violent character and of rapid onset, or the grind of continued worry; falls, blows, fractures, railway and other accidents, and surgical operations; lowered physical states of health; pregnancy, parturition, and lactation; and other mental affections such as mania and melancholia. It is to be observed that the causation of mental confusion is identical with that of nerve exhaustion, or, to use a word which is singularly imprecise, neurasthenia. It is, indeed, difficult to regard the symptoms of confusional insanity as other than further developments of the symptoms of nerve exhaustion. A distinction has been arbitrarily drawn, chiefly, it may be thought, for the reason that "nerve exhaustion" has been for the most part treated outside asylums, while the severer symptoms of "mental confusion" have appeared to necessitate certification and a sojourn in an asylum.

Symptomatology.—Description of the symptoms of mental confusion conveniently begins where description of the symptoms of nerve exhaustion leaves off. The headache, speedy onset of fatigue on slight exertion or weariness without preceding exertion, insomnia, loss of flesh, and other somatic symptoms are accentuated. Speech becomes slow, and is suggestive of sluggishness of the stream of mental associations. There is evidently some degree of obfuscation, and the patient is slow to take in impressions and to convert them into perceptions. Gradually there appears an imperfect and perhaps perverted recognition of persons and things, so that the patient finds it more and more difficult to determine where

he really is in space, at what point of time he in fact is, and, perhaps even who he is: he is said to be disorientated in space, in time, and in personality. The power of fixation in memory becomes poor, and may be so bad that he forgets events which have just occurred, or, indeed, forgets them as rapidly as they occur. A muddling of past memories also occurs, and in some cases there is a complete loss in the power of reproducing past events. Judgment is diminished, and delusions readily form. Affectivity is also diminished: the face is expressionless, and the patient seems to be without feelings or desires. Such defects in the various mental processes may amount to complete inertia and cessation of all mental operations—that is, to a stupor in which no stimulation produces any reaction.

As a variation from this set of symptoms in which inertia and stupor are the outstanding results of intoxication or exhaustion, or both, the patient may be restless and aimlessly wander from place to place in his room or house or in the outside world. It would appear likely that in many cases, while the activities of the highest centres are diminished or inhibited, those of the lower and more automatic are in consequence augmented. In such cases, sets of ideas and memories, seemingly uncorrelated, play a part, much as in a dream. The patient behaves as if acting in some imaginary environment and, according to the character and setting of the hallucinations, is agitated, excited or pleased, or displays some other appropriate reaction.

The chief points of distinction between *mania* and *confusion* may be conveniently indicated here. In confusion, perception of surroundings and of persons about the patient is indistinct and muddled: in mania, perception is very keen and rapid, and there is but little that escapes the patient's notice. In mania there is a rapid passage of ideas whose connexion is often difficult to follow; nevertheless on analysis the associations will be found to be due to the memory or to the reason, while associations of assonance, of rhymes, of analogy, and of contrast may also be traced: in confusion, ideas are unconnected with each other or with the environment. On recovery, the patient who has suffered from mania remembers the course of his illness: in confusion the recollection, if any, is imperfect, and as of an ill-remembered dream.

With the mental symptoms of confusional insanity are combined some which are somatic

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and suggest debility. The patient has lost flesh and is anæmic; the pulse may be rapid, small, compressible, and sometimes irregular, and there may be a diminution in volume of the first sound of the heart; the extremities may be cyanosed and cold. Appetite is lost, the tongue is furred, the breath and the stools are apt to be foul, and the bowels are constipated. Occasionally there is evidence of dilatation of the stomach. The amount of the urine and of the contained chlorides and phosphates is diminished during the attack and increased at its end. The presence of albumin, indican, acetones, diacetic and oxybutyric acids has been noticed in certain cases. So far as the nervous system is concerned, inequality of the pupils is sometimes present, while reaction to light and accommodation are, though usually normal, occasionally retarded. Tendon reflexes vary, but for the most part they are exaggerated. Speech is sometimes sluggish and hesitating, and the patient obviously finds difficulty in suiting words to ideas.

The course of a case of acute confusion is very variable. The onset may be slow and insidious, or almost sudden. Symptoms may last from a period of a few days to years, and as a rule they clear up, but gradually and slowly. When the somatic symptoms definitely improve, but hebétude, amnesia, and disorientation persist, chronicity may be regarded as probable. In most cases, however, the prognosis is good. Death occasionally occurs from exhaustion, or from the severity of the action of the intoxicating agent.

Acute delirious mania or acute confusional insanity (Bell's delirium).—The group of symptoms known under these names is the manifestation of a general infection. It may occur associated with some recognized infection, as enteric fever, or with one unknown. The symptoms are those of the confusional state above described, but are of extreme severity. The initial headache, malaise, and depression are great, and are soon succeeded by restlessness and agitation, which become intense and violent and suggest a state of mind of the utmost excitement and anger or terror, or both. The temperature is raised, and the pulse and respiration rates are high. The symptoms and signs of the "typhoid state" soon develop, and death is the usual termination.

Morbid anatomy.—The structural changes which have been described in cases of confusion are exactly those common to all intoxications

particularly affecting the nervous system; they consist essentially in swelling, deformity, and chromatolysis of the cells of the cortex. In acute delirious mania, in which the alterations are most marked, there are cerebral hyperæmia, venous and lymphatic engorgement, œdema of the convolutions, meningeal injection, and extravasations of blood, both meningeal and cortical. About the vessels are extravasations of round cells. The cortical cells are as above described, and their prolongations exhibit varicosities. The lesions, in short, are those of meningo-encephalitis.

Treatment.—Bearing in mind the causation of confusional insanity, the indications for treatment lie in endeavouring to counteract the effects of the toxic or exhausting agent. The patient should be put to bed, and so far as possible the perceptive field should be diminished by limiting stimuli from without. This is best affected by isolation, so that none but the patient's professional attendants see him, while the ministrations of these should be carried on with a minimum of conversation, fuss, or vexatious and unnecessary interference with the patient's wishes. Patients, though restless, can often be persuaded to stay in bed, but if they insist on getting up it is better to allow them to roam around their rooms than to struggle to keep them in bed or to use strait-jackets or other means of mechanical restraint. Justifiable restraint consists in preventing the patient from damaging or killing himself or assaulting others. If possible, the bed should be in the open air. If this is not practicable, the room should be amply ventilated. Only too often the atmosphere of the strong-rooms, padded rooms, and other places where such patients are tended is vitiated and noisome, and tends to increase restlessness and agitation. The alimentary tract should be zealously attended to; in the early stages it is probable that drastic purgatives and enemata may have to be used, regular evacuation being afterwards assured by the daily administration of some efficient aperient. Food should be given in large quantities, and it will at first probably have to be of a liquid sort, for solid food is usually refused. Warm baths sometimes allay excitement and induce sleep. Hypnotics or sedatives may be administered where indicated. In some cases such drugs appear to increase excitement and sleeplessness. The dose required varies very much with different individuals; some patients only react with very small doses, others only

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with large. When improvement sets in, relaxation in treatment should lag far behind. Relapses are often induced by undue advantage being taken of an abatement of symptoms. Convalescence should be prolonged, and every effort should be made to raise the level of the patient's state of general nutrition to a high standard.

TRAUMATIC PSYCHOSIS

Whether or no trauma—somatic or psychic—produces auto-intoxication is at present a moot point, but there is at least no doubt that the mental symptoms associated with it are, in most cases, those of confusion. In some cases the symptoms apparently induced by trauma occur in individuals who suffer from renal or cardio-vascular or gastro-intestinal insufficiency: it is not improbable that in such there exists a particular liability to intoxication.

Symptoms may come on directly after the trauma or shock, or they may be postponed from ten to fourteen days, or, indeed, may not appear for weeks or months. In the last instance they are probably only secondarily traumatic, and are induced rather by the exhaustion of inanition, or of worry or pain or suppuration, than by the trauma itself. Early symptoms may consist in modifications of character, irritability, gloom, want of will-power, obfuscation, torpor, stupor, or automatism. These may later be complicated by failure of judgment and delusions, by excitement and agitation, and by hallucinations. The syndrome is, in fact, that of confusional insanity.

INFECTIVE PSYCHOSIS

The mental symptoms which attend infective processes may appear concurrently with the somatic, and indeed in some cases precede them; or they may come on when the fever and other symptoms are at their height; or, lastly, they may be delayed until the fever has abated and convalescence seems about to be established.

The **symptoms**, whenever they appear, are those of confusion, and at the commencement of the illness or during its height are usually those of acute delirium, and may even amount in severity to those of acute delirious mania. This naturally renders nursing extraordinarily difficult, since the patient may constantly strive to escape the scenes of hallucinatory horror in which he finds himself, and may fight those about him, or try to force his way through doors and windows. The symptoms

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which come on after the acute stage of an infection is over are more obviously those of exhaustion or asthenia, and include blunting of intellectual activity, in some cases amounting even to stupidity, and perhaps restlessness and agitation.

In acute cases the **anatomical lesions** have been found to be those of meningo-encephalitis, with more or less hyperæmia, œdema, and alterations in cells and fibres.

Prognosis and treatment.—The prognosis is, in general, good. In some cases the patient remains, on recovery from the acute symptoms, at a lower mental level than the one usual to him before his illness. Death may occur from acute toxæmia. Treatment is precisely that for confusional insanity due to any other cause, except in so far as the somatic symptoms of the infective process are amenable to some special procedure.

E. D. MACNAMARA.

CONGELATION (*see* CARBON DIOXIDE SNOW, TREATMENT BY).

CONGENITAL FAMILY CHOLÆMIA (*see* JAUNDICE).

CONGENITAL HEART DISEASE (*see* HEART, CONGENITAL DISEASE OF).

CONGENITAL HYPERTROPHIC STENOSIS OF PYLORUS (*see* PYLORUS, CONGENITAL HYPERTROPHIC STENOSIS OF).

CONGENITAL LARYNGEAL STRIDOR (*see* STRIDOR).

CONGENITAL SYPHILIS (*see* SYPHILIS).

CONJUNCTIVITIS.—An inflammation of the conjunctiva which may affect both the palpebral and ocular layers, either together or separately. The different varieties can be recognized clinically according to the severity of the inflammatory reaction or its distribution.

The visible **signs** of conjunctivitis are:

(1) General or localized injection of the blood-vessels contained in the conjunctiva which lines the inner surface of the lids and the eyeball. This, in acute cases, gives to the sclerotic an intensely red appearance, while the accompanying cellular infiltration of the mucous membrane causes swelling, variable in amount, but sufficient to mask almost entirely the individual vessels so plainly visible in the normal healthy conjunctiva. In more chronic cases the vessels are merely dilated or tortuous, either generally or locally.

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(2) Fluid secretion from the conjunctiva, producing a mucoid, muco-purulent, or purulent discharge that exudes from the palpebral aperture during the day and glues the edges of the lids together at night, so that in the morning the lids cannot be opened until the dried-up discharge has been washed away.

These two signs in a greater or less degree characterize all varieties of conjunctivitis. Both eyes are usually affected, one shortly before the other.

The **symptoms** complained of may be irritation, a general feeling of discomfort, itching, or a sensation of sand in the eyes, but there is no pain or photophobia, and the cornea is always bright and clear. These latter symptoms are never found in a purely uncomplicated conjunctivitis, and their presence always indicates extension, however slight, of the inflammation from the conjunctiva to the cornea. One should always be suspicious of a conjunctivitis which remains monocular or localized to some particular part of the conjunctiva for any length of time, and defies all the ordinary methods of treatment. There is probably some unusual or atypical form of conjunctivitis present—irritation of a foreign body hidden away somewhere (e.g. hair in the punctum, ingrowing lash, conjunctival growth), or some superficial ulceration of the cornea of a very transitory yet recurrent nature. In these circumstances a very careful search should be made by eversion of the lids, examination with a magnifying lens, etc., to ascertain the cause.

The following varieties of **acute** conjunctivitis are those most commonly met with:—

1. CATARRHAL CONJUNCTIVITIS

In this variety the whole eyeball looks intensely injected, and the blood-vessels are sometimes so engorged that some of them give way and produce small characteristic subconjunctival hæmorrhages. The inner surface of the lids is also considerably swollen, but no definite elevations are seen. The discharge is profuse, and usually of a mucoid nature, though in severe cases it may be muco-purulent. Sometimes there are small white patches at several points round the limbus resembling phlyctenules, but, although pathologically indistinguishable from the latter, they are clinically entirely different, are due to direct infection from the lids, and will disappear as soon as the conjunctivitis is cured. If the practitioner makes a diagnosis of phlyctenular conjunctivitis his prognosis must be influenced,

since the latter affection may be of longer duration and, as will be described later on, is dependent upon the state of the general health.

Catarrhal conjunctivitis is caused by the activity of the Koch-Weeks bacillus, an organism which is not unlike the influenza bacillus and can be demonstrated in smears made from the discharge.

Prognosis and treatment.—Although the condition to all outward appearance looks to the patient rather alarming, it is by no means difficult to treat; even the ordinary method of cleanliness will often bring about a cure in a week or ten days, but it must be remembered that the affection is very contagious, and, unless precautions are carefully taken in regard to towels, handkerchiefs, etc., will spread rapidly from one member of a family to another until the whole household becomes affected.

Treatment, as in all cases of conjunctivitis, consists in washing out the conjunctival sac with a boric-acid (10 gr. to the ounce) or perchloride-of-mercury (1 in 5,000–8,000) lotion by means of a glass syringe, eyebath, or other form of receptacle such as the undine, or a small jug with a good spout; this should be repeated several times in the day. At night some lubricant, e.g. boric ointment or vaselin, should be well smeared along the edge of the lids to prevent the adhesion which would otherwise take place during sleep. The neglect of this precaution often causes delay in recovery, since the organisms grow rapidly while the lids are closed and continue to reinfect the conjunctiva each day.

No complications, as a rule, are to be anticipated, and if the treatment indicated is followed a cure can confidently be expected in a week or two, and almost certainly within three weeks. If there is any tendency for recovery to be prolonged, it is a good plan to paint the lids once or twice a week with silver nitrate (10 gr. to the ounce) or protargol (20 or 30 per cent.) in the same way as is described under Purulent Conjunctivitis (*see below*). An excellent alternative as a lotion in this and any other form of conjunctivitis is hydrochlorate of quinine (3 or 4 gr. to the ounce).

2. PURULENT CONJUNCTIVITIS

This form is represented by the well-known clinical types of gonorrhœal ophthalmia and ophthalmia neonatorum, the former in adults, the latter in new-born babies, and differs from other forms in the abundance and consistence of the discharge as well as the possibility of

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secondary implication of the cornea. After a short period of incubation, varying from a few hours to a few days (three days in ophthalmia neonatorum), the conjunctiva becomes intensely inflamed and swollen by œdema (chemosis). This is quickly followed by an abundant muco-purulent discharge. The inflammatory process may rapidly spread to the cornea, leading to ulceration and perforation in the course of a few days, and resulting in prolapse of the iris into the wound with all the attendant risks and dangers.

In purulent conjunctivitis, due to any cause, the preauricular gland is generally found to be enlarged, an indication of the virulence of the inflammation.

The profuse white discharge welling up from beneath the lids as soon as they are separated is characteristic, and must be thoroughly cleared away before any proper examination of the conjunctiva and cornea can be made. It is highly important that an opinion regarding the condition of the latter should be formed at the earliest possible moment.

Obviously, this is an extremely dangerous type of conjunctivitis and, especially in adults, may cause total destruction of the eye in the course of a few days. It is also very contagious, so that, when one eye alone is affected, vigorous efforts should be directed towards shutting off from the unaffected eye any channel of infection. To accomplish this, a Buller's shield should be carefully be applied over the normal eye and secured in position by strapping, special attention being paid to the nasal boundary of the orbit; to prevent moisture from collecting and obscuring the glass, it is advisable to have a small piece of drainage-tube inserted beneath it on the temporal side to form a communication with the external air. Protecting goggles should be worn by those in attendance on the patient, and scrupulous care exercised in washing the hands afterwards.

Treatment.—In new-born children the percentage of cases of ophthalmia neonatorum has been greatly diminished by the adoption of Credé's method of instilling a few drops of a 2-per-cent. silver-nitrate solution into the baby's eyes as soon as it is born, but it must not be assumed on this account that silver-nitrate drops can be used indiscriminately by unskilled persons for a long time. The conjunctiva and cornea tolerate the occasional use of silver, but its prolonged use leads to

permanent staining of the former and ulceration of the latter.

The object aimed at is to lessen the amount of discharge and control the growth of the organisms which prove so disastrous to the cornea. Lotions must be used as in all forms of conjunctivitis, but no antiseptic fluid can be applied to the eye strong enough to destroy the organisms without affecting adversely the delicate structures of the eyeball itself. Many drugs, however, can be used in sufficiently strong solutions to inhibit growth, and if poured into the eye from a definite height, either out of an undine or a small jug, the organisms are mechanically washed out with the discharge. If the washing out is repeated often enough, an appreciable control is established over the number of organisms in the conjunctival sac. The aim should, therefore, be to secure continuous irrigation as nearly as possible; it is generally found that hourly washing out in the day, and four-hourly during the night, is sufficiently often, and compatible with the requirements of sleep. The lotion generally used is perchloride of mercury (1 in 5,000 or 7,000) or eusol (1 in 6).

If there is much chemosis it will often be found helpful to apply iced compresses in the intervals between the douchings, as the œdema may be sufficiently severe to obstruct mechanically the circulation at the limbus and so cause necrosis of the cornea; but this treatment keeps the eyes closed, which is a distinct disadvantage. A daily examination of the cornea should be made as long as the symptoms are acute, and if any difficulty is experienced in separating the lids for this purpose, lid retractors must be used, since the cornea may easily be damaged by the careless use of the fingers. An ulcer on the verge of perforation may in a moment be converted into an actual perforation, and a cornea which up to that time was healthy may be scratched, when the abrasion will be quickly infected, and an ulcer result.

In addition to the use of lotions, the palpebral conjunctiva, after eversion of the lids (see EYE, EXAMINATION OF), must be painted once a day with some preparation of silver, the strongest one being silver nitrate (10 gr. to the ounce); protargol (15-30 per cent.) and argyrol of the same strength are both weaker than the silver nitrate. This painting is best carried out by means of a fine cotton-wool swab on the end of a glass rod; as the wool can be burnt and the glass rod boiled, this method is far

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preferable to a camel's-hair brush. With regard to this procedure, a few words of caution are desirable: never use stronger solutions of silver nitrate than 2 per cent. (or 10 gr. to the ounce), never paint the lids more than once a day, and never use the strongest preparations in the absence of discharge. It is owing to neglect of these precautions that disasters often occur.

All practitioners are familiar with the use of silver nitrate in the treatment of severe forms of ophthalmia, but in their anxiety to effect a rapid cure they often apply it too frequently or with too much vigour, with the result that the caustic action often extends more deeply than is necessary and, by its action on the cornea, actually produces the very condition the treatment is intended to avoid. The mischief thus caused is mistaken for damage produced by the original infection, and so renewed efforts are made with the silver nitrate, only to be followed by further destruction of corneal tissue. As soon as the discharge becomes less profuse, some milder preparations, such as protargol, in varying strengths, should be substituted for the silver nitrate, and in this way the effects of over-zealous treatment can be avoided. I know of no class of case which responds more readily to a change of treatment than bad cases of gonorrhœal ophthalmia.

The proper method of painting the lids is as follows: Take up a position behind the patient, who will be seated in a chair or lying on a table or couch; if a baby, it is best to hold the child's head gently between the knees while its feet rest on the mother's or nurse's lap. Evert the upper lid in an adult as described under EYE, EXAMINATION OF, and in babies by gently separating the lids with the finger and thumb placed close together at the outer canthus, when they will generally turn out. Dry the surface of the conjunctiva with a small cotton-wool sponge wrung out of boric lotion, and apply the silver nitrate by rubbing over the conjunctiva with a cotton-wool swab on the end of a glass rod, dipped in the solution. After a few seconds the conjunctiva becomes bleached, showing that the silver has acted by destroying the epithelium. Now mop with a wool sponge any superfluous fluid that may run over the lids. The lids can then be replaced.

Should the cornea become affected, as manifested by a general grey haze over the surface, atropine must be instilled immediately, in the form either of ointment or of drops (1 per cent.), three times a day, and the pupil kept fully

dilated. This tends to prevent adhesion of the iris to the cornea in the event of perforation, keeps the eyes as far as possible at rest, and promotes healing. Continue to treat the conjunctiva at the same time.

The **prognosis** depends upon the extent of this complication, but in any case several weeks must elapse before a definite pronouncement can be made with regard to the completion of the cure; bacteriological examinations should be made periodically until all signs of the gonococcus have been eliminated.

Even when corneal complications arise, it is astonishing how much better, in children, is the ultimate result than could have been expected. In adults, however, the result is far less favourable, and the eye is very often lost.

Since purulent conjunctivitis from any cause produces considerable depression in the general health, tonics, fresh air, etc., should be prescribed.

3. MEMBRANOUS OR DIPHThERIC CONJUNCTIVITIS

Characteristically this is an acute conjunctivitis due to the Klebs-Löffler bacillus, but a very similar condition is sometimes found to be due to streptococcal infection. The essential features of this form are the greyish colour of the swollen conjunctiva and the subsequent appearance on the conjunctival surface of the lids of a definite membrane which, if peeled off, leaves a raw bleeding surface, just as is seen in the corresponding condition affecting the larynx. There is often some brawny swelling of the conjunctiva and lids, with a certain amount of purulent discharge, though not so profuse as in the gonorrhœal type. The general health is much affected, as shown by general depression and rise of temperature.

The cornea is likely to be involved, and the lids may become adherent to the globe through the union of the conjunctival surfaces (symblepharon); this can be prevented by passing a glass rod daily between the lid and the globe so that the adhesions are broken down wherever they form.

The **prognosis** is favourable provided that the case is treated at once and energetic steps are taken to avoid complications.

Treatment.—The diagnosis having been definitely established by bacteriological investigation, antitoxin treatment must at once be instituted, together with the local treatment described under Purulent Conjunctivitis, with the single exception that the lids must never

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be painted with any silver preparation, but the membrane left to exfoliate.

The general health must carefully be attended to and the same precautions adopted regarding contagion as in other cases of diphtheria.

4. PHLYCTENULAR CONJUNCTIVITIS

This form of acute conjunctivitis is found almost exclusively in children. It is characterized by localized areas of conjunctival injection at various points on the limbus, surrounding little raised spots visible to the naked eye and consisting of collections of mononuclear cells in the adenoid and subepithelial layers. When the epithelium over the little swellings breaks down they form small ulcers which, however, do not give rise to photophobia unless the cornea is involved. The condition is due to auto-intoxication from some remote focus, generally tuberculous, and is encouraged by malnutrition due to bad hygienic surroundings; for this reason it is most commonly met with in children of the poor, and is of long duration, or frequent recurrence, often persisting for several months or even years.

The local **symptoms** are very slight, comprising only those of conjunctivitis in a modified form; there is no photophobia, unless the inflammation spreads to the cornea, a very likely contingency. The patients are generally subjects of glandular enlargement of the neck, and it has been said that removal of the glands checks the course of the affection in the eye.

Diagnosis is not difficult if the points already described are borne in mind. In adults the condition is most likely to be mistaken for *episcleritis*, which runs a much more chronic course and does not yield readily to treatment. The patch of injection at the limbus is common to both, but in *episcleritis* the redness is of a deeper and pinker shade; it tends also to spread to the ciliary body rather than to the cornea, as is the rule in phlyctenular conjunctivitis. Moreover, *episcleritis* takes a long time before it is completely cured, whereas an uncomplicated phlyctenular conjunctivitis gets well in a few weeks under suitable treatment, provided no corneal complications arise.

Treatment consists in washing out the eye two or three times a day with some lotion, usually perchloride of mercury (1 in 7,000), followed by gentle massage of the globe by means of the closed lid after introduction of some ointment, e.g. boric-acid; yellow oxide of mercury, advocated in most books,

is quite unnecessary, and often aggravates the injection and irritation, especially when there happens to be any corneal ulceration. The above method of treatment will often bring about a cure in a week or so, whereas my experience is that the use of yellow oxide sometimes actually keeps up the inflammation.

Tonics, and especially change of air to the seaside, will hasten recovery and diminish the tendency to relapse.

The **chronic** forms of conjunctivitis most commonly met with are the following:—

1. ANGULAR CONJUNCTIVITIS

This, as the name implies, occurs at the inner and outer canthi, and is accompanied by some injection of the conjunctiva along the edges of the lids. It is essentially an inflammation of the palpebral layer, the ocular portion never being affected. The redness extends over the edges of the lids on to the skin surface, causing a certain amount of excoriation at the outer and inner canthi.

Etiology.—The condition is due to a specific organism, the Morax-Axenfeld bacillus, a small rod-shaped diplobacillus which can very easily be stained and demonstrated in a smear from the conjunctival secretion.

The **signs and symptoms** are much the same as in the acute forms, but are of a much slighter degree. There is very little discharge, but just enough to glue the edges of the lids together at night. The patient complains of a considerable amount of irritation and heaviness of the lids; and especially of an itching sensation which is, I think, peculiar to the angular variety. The symptoms are not severe enough to lead the patient to seek advice early, and when the case first comes under observation the condition has usually existed for three or four weeks, this relatively long history being a very suggestive element in the diagnosis.

The **prognosis** is distinctly favourable, though it may take from three to six weeks to effect a complete cure, and the affection is very liable to recur. Unlike acute catarrhal conjunctivitis, it never gets well spontaneously, nor with boric-acid or perchloride-of-mercury lotions, which often have already been tried by the patient on his own account.

The proper **treatment** consists in washing out the eye with a lotion containing zinc sulphate or zinc chloride (1-3 gr. to the ounce). The zinc salts are an absolute specific for the Morax-Axenfeld bacillus, and the inflammation

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quickly subsides under their influence ; in two or three weeks it has generally disappeared, though it is best to persevere with the treatment for two or three weeks longer to prevent recurrence. It is better to begin with the strength of 1 gr. to the ounce, as some patients are very sensitive to the astringent action of this drug and cannot bear a stronger solution ; in these circumstances the weaker lotion is just as efficient, and the strength can easily be increased if necessary. A good alternative to zinc is alum (4 gr. to the ounce), but it is a weaker preparation. As the lids, to a certain extent, stick together at night, before going to sleep some ointment must be spread along their edges.

If an angular or chronic conjunctivitis is slow in responding to the foregoing treatment, the conjunctiva may be painted with weak silver solutions (e.g. protargol 15 or 20 per cent.) or with silver nitrate (1 per cent.) once or twice a week, but not so often as in the acute forms. Since smoke, dust, bad air, late hours, reading by artificial light, and small errors of refraction tending to produce eyestrain aggravate the symptoms, the treatment must include attention to these details.

2. FOLLICULAR CONJUNCTIVITIS

A chronic form of conjunctivitis associated with overgrowth of the follicular tissue in the loose portion of the conjunctiva, giving rise to pale translucent elevations in the fornices. These are always seen in two or more rows parallel to the edges of the lids, and are never found on the conjunctiva covering the tarsal plate, thus differing from those of trachoma. The condition is comparable to enlargements of the tonsils, and is often found in children who show a tendency to adenoid-tissue overgrowth.

The **symptoms** are much the same as in other varieties of chronic conjunctivitis, but are much milder ; indeed, the secretion is seldom sufficient to produce sticking of the lids at night.

The best **treatment** consists in the application of fairly strong astringent lotions or in painting the follicles with alum stick. There is no necessity for such drastic measures as expression, or painting with silver nitrate, which, though they lead to disappearance of the follicles, are needlessly violent.

3. TRACHOMA (GRANULAR CONJUNCTIVITIS)

This form is endemic in certain places, and has found its way into England since the ad-

mission of aliens—e.g. Russian Jews—into the country. But it is by no means so common as it was several years ago, the introduction of more careful supervision in the examination of the eyes of aliens landing at the ports having eliminated a considerable number of cases.

The disease is subacute and very persistent. It is distinguished from all other forms of conjunctivitis by the presence of grey translucent gelatinous granulations like sago grains scattered uniformly all over the palpebral conjunctiva, and not specially confined to the fornices. The granulations consist of collections of leucocytes and lymphocytes, caused no doubt by the presence of the trachoma organism (not yet discovered) in the subconjunctival tissue ; these collections push the epithelium in front of them and give rise to numerous elevations over the inner surface of the lids. The cornea soon becomes secondarily infected at the upper part, where the upper lid is in contact with it, and granulation tissue rich in blood-vessels grows into the cornea beneath the epithelium and superficially to Bowman's membrane, producing a hazy infiltration in the affected area ; this is known as trachomatous pannus. At this stage obstinate photophobia may develop and, in the absence of treatment, ulceration of the cornea may occur and lead eventually to perforation and destruction of the eye.

Diagnosis.—When the disease presents the typical appearance just described, there is little fear of mistaking it for anything else, but when there are only a few granulations confined to the fornix, as in early stages, there is some likelihood of confusing it with *follicular* conjunctivitis. Doubtful cases should be watched carefully for a few weeks ; if trachoma is present, granulations are sure to appear on the palpebral conjunctiva over the tarsal plate while the cornea becomes roughened at the upper quadrant. In follicular conjunctivitis the follicles remain confined to the fornix, are never found on the upper lid, and do not affect the cornea.

Various forms of **treatment** have been employed in trachoma, all of which are directed to destroying the granulations and as much of the deeper layers of the conjunctiva in which they grow as is safe. Opinions only differ as to which is the most effective method. In the early stages it is a good plan to express the granulations by means of special forceps applied to the everted lid—a procedure which must be carried out under an anæsthetic, as it is

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extremely painful. This is followed by painting with strong perchloride of mercury (1 in 200); much of the conjunctival tissue is thus destroyed, together with the granulations in all stages of development. Another method is to scarify the conjunctiva with a hard toothbrush, and afterwards to paint the lids in the same way.

Whatever method is adopted at first, daily painting with silver nitrate (2 per cent.) must be the routine treatment, and later on, when the condition becomes more chronic, copper-sulphate stick should be used instead. As long as there is much discharge, copper should not, as a rule, be applied.

Another very useful procedure is to hold a stick of CO₂ snow in contact with the conjunctival surface of the lid for about half a minute. If employed once a week for a few weeks, this is one of the quickest and most successful methods of treatment, but, owing to the thawing of the parts which are not destroyed, it is one of the most painful, often keeping the patient awake for a night or two afterwards; this is more than some patients can bear.

In the intervals between the various applications the eyes must be washed out with perchloride-of-mercury lotion (1 in 5,000), and a little ointment spread along the lids at night, atropine being also instilled as soon as the cornea becomes affected. A disease so destructive to the conjunctival tissue leads to much scarring and cicatricial contraction of the conjunctiva and lids, so that even after a cure has been obtained the lids are often distorted, leading to entropion, trichiasis, and ptosis; permanent scars may persist on the cornea. At the same time the various secretory glands are destroyed, leading to dryness of the conjunctiva and eventually to xerosis.

4. SPRING CATARRH

A rare condition occurring in the summer months. Characteristically, it produces flat-topped elevations on the conjunctival surface of the upper lid, accompanied by a milky discharge; a superabundance of eosinophil leucocytes is found in the secretion. It has sometimes been mistaken for trachoma, but the individual elevations are larger and flatter, are much more regularly arranged, being grouped together like pavement stones, and occur only on the tarsal plate.

In another form, localized vascular outgrowths appear on the eyeball at the limbus,

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but it is not easy to diagnose, and is so rare that it need not be considered here.

Treatment consists in applying a lotion composed of dilute acetic acid (3 min. to the ounce) combined with adrenalin (1½ dr. of 1-in-1,000 to the ounce); the ordinary lotions have no effect whatever.

OTHER AFFECTIONS OF THE CONJUNCTIVA

Such affections as *concretions* (chalky deposits on the surface of the mucous membrane), *pinguicula* (yellow elastic tissue), situated on each side of the globe in that part of it exposed by the palpebral aperture, and *pterygium* (encroachment of the conjunctiva on to the cornea), situated in the same region as pinguicula, require no treatment as a rule, unless they cause trouble, when operative measures may be called for. It is well, however, to bear in mind that almost any form of chronic conjunctivitis or conjunctival overgrowth is benefited by the use of a zinc lotion combined with adrenalin.

MALCOLM L. HEPBURN.

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Etiology and pathology.—Constipation may be due to (1) the passage through the colon being delayed, whilst defæcation is normal—*colic constipation*; (2) the evacuation from the pelvic colon being inadequately performed, whilst the passage through the colon is normal—*dyschezia*; (3) insufficient formation of fæces.

1. **Colic constipation.**—Delay in the passage of fæces through the intestines is due (a) to their motor activity being deficient, or (b) to the force required to carry the fæces to the pelvic colon being excessive. (a) *Defective motor activity of the colon* may result from

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weakness of its musculature, deficient reflex activity, inhibition, or uncontrolled and irregular action. Weakness of the musculature may be due to congenital hypoplasia, which is the cause of the constitutional constipation from which several members of a family may suffer; to acquired hypoplasia, which is the cause of senile constipation and the constipation which may follow such local diseases as dysentery; or to functional insufficiency without structural change, as in chlorosis, acute fevers, and the cachexia of cancer and tuberculosis. The reflexes which maintain intestinal activity may be deficient owing to the stimulation being too weak on account of want of exercise or lack of mechanical and chemical stimulants in the food. They are also deficient when the excitability of the mucous membrane is impaired as a result of catarrh and of long-continued irritation by purgatives. Constipation is a very frequent symptom of neurasthenia, the depressed condition of the nervous system leading to a diminished response to the stimuli which normally excite intestinal activity. The inhibitory sympathetic nerves may be stimulated directly in lead poisoning, centrally by depressing emotions, and reflexly in painful diseases and injuries of any part of the body, but particularly of the abdominal and pelvic viscera.

Constipation may result from painful spasm, induced reflexly by the presence of an irritant in the colon of a patient with an abnormally excitable nervous system (*enterospasm, spastic constipation*). Such patients are generally neurasthenic, and there is often a history of worry or overwork immediately preceding an attack. The irritant which gives rise to the exaggerated reflexes is in most cases hard faeces, the retention resulting from the ordinary neurasthenic constipation, which is generally present in the intervals between the attacks. Similar spasmodic contractions of the intestines occur as a result of excessive smoking and in lead poisoning.

(b) *The work of the intestinal musculature is excessive* when the consistence of the faeces offers more than the normal degree of resistance, owing to insufficient consumption of water or its excessive loss in the urine or sweat, and when there is a diminution in the intestinal lumen owing to organic stricture or pressure exerted from without.

2. **Dyschezia.**—Dyschezia is due to a want of proper proportion between the power of expelling the faeces from the pelvic colon and

rectum and the force required to do this completely. It may therefore result from inefficient defaecation, or from an obstacle to efficient defaecation, such as abnormally hard and bulky faeces, and functional and organic strictures of the rectum and anal canal. Dyschezia due to *inefficient defaecation* is most commonly the result of disregarding the call to defaecation from ignorance or laziness, or from fear of pain in diseases of the anus and the neighbouring organs. If the call to defaecation is disregarded, the sensation of fullness in the rectum passes off owing to relaxation of the tonic contraction of its muscular coat. If the call is again disregarded after its return on the arrival of more faeces in the rectum, further relaxation occurs. More and more faeces accumulate in the rectum, the muscular coat of which becomes more and more relaxed. As the force required to empty the rectum when over-distended with faeces is much greater than that required under normal conditions, evacuation is now likely to be incomplete, even if a great effort be made, and it may finally become impossible without mechanical aid; consequently faeces are always present in the rectum instead of only for a few minutes before defaecation, and the lumen of the rectum is permanently increased owing to its atonic condition.

Dyschezia may be due to various other causes, such as weakness of the voluntary muscles of defaecation, the assumption of an unsuitable position during defaecation, and hysteria. But whatever the primary cause, the final result is the same. The incomplete evacuation of the rectum results in an accumulation of faeces and consequent atony and dilatation of the rectum.

Congenital deficiency of the muscle-sense of the rectum is the cause of the dyschezia of infants, in whom the slight additional distension produced by the introduction of a finger or a piece of soap into the rectum results in an adequate stimulus. In the majority of cases the muscle-sense develops when the infant grows older, but this is occasionally the starting-point of dyschezia which lasts through life. The rectal muscle-sense is abolished or defective in diseases of the spinal cord, in which the defaecation centre itself or the fibres connecting it with the brain are involved.

3. **Insufficient formation of faeces.**—The third great class of constipation is due to the quantity of faeces formed being insufficient to produce an adequate stimulus in the pelvic

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colon and rectum, and to a less extent in the rest of the colon. The insufficient bulk of fæces is due to an inadequate quantity of food residue reaching the colon as a result of anorexia or of œsophageal or pyloric obstruction, or of excessive digestion of food, the result of a "greedy colon."

Symptomatology.—Many people regard themselves as ill if they do not have one action of the bowels a day, but this is really nothing more than a question of convenience, being found to suit the habits and diet of the majority of civilized people. It is, indeed, not very rare to find persons in perfect health who defæcate regularly two or three times a day, and others who obtain an evacuation only once in two, three, or more days, without suffering the smallest ill effects. Such persons, so long as defæcation, when it does occur, is complete, can no more be regarded as diseased than those otherwise normal people whose hearts beat only forty or fifty times a minute. The majority, however, although they may suffer no inconvenience for a considerable time, finally develop symptoms due to fæcal accumulation, gradually increasing quantities of fæces being retained. For practical purposes, therefore, a person may be considered constipated if his bowels are not opened at least once in every forty-eight hours. A less frequently recognized variety of constipation is that known as "cumulative constipation," in which insufficient fæces are excreted although the bowels may be opened every day, a condition analogous to retention of urine with overflow. Lastly, the amount of fæces formed may be normal and the bowels may be opened daily, yet the fæces are abnormally hard and dry owing to prolonged retention before excretion.

It is important to distinguish the symptoms of constipation from those of the conditions, such as neurasthenia and chlorosis, which may give rise to it. There is no doubt that neurasthenia, insanity, epilepsy, asthma, nephritis, diabetes, chlorosis, painful pelvic disorders, and many other diseases may be greatly aggravated by constipation. Apart from these conditions, headache, mental and physical fatigue, vertigo, anorexia, and pigmentation of the skin may result directly from the intestinal intoxication and the reflex effects of constipation. Fæcal retention is a common cause of intestinal flatulence and colic. In rare cases a fæcal tumour and fæcal obstruction occur. A fæcal accumulation in the rectum may cause hæmorrhoids, pruritus ani, catarrhal and

ulcerative proctitis, and neuralgic pains in the back, down the legs, and in the female pelvic organs. Hard fæces make defæcation painful, and their passage may give rise to anal ulcers. Under certain conditions, especially in women, bacteriuria and pyelitis may develop, and constipation may be one factor in the production of muscular rheumatism and of rheumatoid arthritis. Stasis in the cæcum may give rise to chronic appendicitis, and constipation is the chief cause of catarrhal and muco-membranous colitis. Finally, constipation is the primary factor in the production of volvulus, and of the diverticula which occur in the colons of elderly people.

Treatment. Hygiene of the bowels.—In no circumstances should the patient fail to make an effort to open his bowels after breakfast, even if he feels no desire to do so, and a call to defæcation felt at any other hour in the day should be obeyed at once, however inconvenient the time. Sufficient time should always be spent over the act of defæcation, and it is often advisable to pay two visits to the closet at short intervals. In order to prevent the temptation to hurry over defæcation the closet should be clean, devoid of smell, and properly warmed in winter. In dyschezia a footstool nine inches lower than the seat should be provided, and in severe cases the patient should crouch over a bed-pan placed on the floor.

Diet.—It is most important to see that enough food is taken, as constipation is often as much due to its insufficient quantity as to its unsuitable quality. The diet should contain an increased proportion of vegetable foods, especially those which contain much cellulose, organic acids, and sugar. Fresh or dried fruit should be taken three times a day, and green vegetables or salad eaten at lunch and dinner. Porridge and cream, wholemeal brown bread, and oatcake are also useful. Care should be taken that sufficient fluid is drunk; a glass of cold water taken on rising in the morning often helps the bowels to act after breakfast.

Drugs.—The majority of cases of constipation can be cured without drugs, if proper treatment is instituted at a sufficiently early stage. In dyschezia purgatives are either absolutely useless or they only have an effect when fluid stools are produced, a considerable quantity of fluid and nutritive material being thereby wasted. In the constipation of curable diseases, such as chlorosis and neurasthenia, and

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of chronic renal diseases, diabetes, and insanity, all of which are aggravated by the constipation which is commonly present, purgatives should be regularly given. They are also useful for making the stools soft when defæcation is painful as a result of inflamed hæmorrhoids or anal ulcer, in diseases of the pelvic organs, and when straining at stool is accompanied by danger, as in patients liable to cerebral hæmorrhage. In those cases of constipation due to other causes, in which non-medical treatment proves insufficient, purgatives must also be used, but an effort should be made to dispense with drugs at the earliest possible moment. The stool produced by an aperient should be normal in size and consistence; the dose should be so regulated that one stool is passed every day, and the desire to defæcate is felt immediately after breakfast. An infusion of senna pods is particularly useful, as the dose can be regulated from day to day by the patient; an attempt should be made at intervals to reduce the number used by one at a time, until finally none may be required. The aperient should cause no pain or discomfort, and should not irritate the intestinal mucous membrane sufficiently to produce any inflammatory change. If it is probable that the purgative will be required permanently, one such as aloes, cascara, or senna should be chosen, which is likely to maintain its good effect without any increase in the dose.

In constipation due to a greedy colon the bulk of the fæces must be increased by the administration of some unirritating substance, such as liquid paraffin or agar-agar, which passes through the intestines without undergoing decomposition or absorption. Liquid paraffin is particularly valuable when the fæces are hard and dry; it is therefore useful in other varieties of constipation besides this, fæces always becoming hard and dry as a result of their abnormal retention in the colon. In dyschezia also the soft stools which result from its use are expelled with less difficulty than ordinary fæces. From 1 dr. to $\frac{1}{2}$ oz. should be taken immediately after meals two or three times a day. A minimum of some essential oil may be added to each dose as a flavouring agent, and in some patients an emulsion must be used, as otherwise some of the paraffin escapes from the rectum with flatus apart from the fæces.

Enemata.—The majority of cases of moderately severe constipation are more or less cumulative, an excess of fæces being always

present in the large intestine. It is therefore necessary that the colon should be evacuated completely before other methods of treatment are adopted. It is generally possible to empty the bowels completely by means of a dose of castor oil, followed perhaps by a saline purge. In severe cases, however, it is necessary to remove the accumulation of fæces from the large intestine by enemata before other treatment is attempted, and in rare instances the rectum can only be emptied by means of the finger.

It is essential in treating dyschezia to keep the rectum and pelvic colon empty, so that they may in time regain their normal tone and contractile power. This can only be accomplished by the use of enemata of water or glycerin every morning if a prolonged attempt to defæcate naturally has proved unsuccessful. The bulk of the water enemata and the strength of the glycerin enemata should be gradually reduced. As a rule, the tone and contractile power slowly return and a cure finally results. In very exceptional cases the atony and paralysis of the rectum are so complete that recovery is impossible; in such cases treatment by enemata, though it does not cure, is the only way in which a regular evacuation can be obtained.

Hydrotherapy.—A cold bath or a cold douche after a hot bath is a very valuable addition to the series of stimuli which leads to the morning evacuation. The spasm in spastic constipation is often benefited by a hot bath; and when constipation is due to some painful pelvic condition, the latter and the associated spasm of the sphincter ani may be relieved by the use of a hot sitz-bath.

Exercise and massage.—Regular exercise in the open air is one of the most important means of preventing constipation, especially in those who follow a sedentary occupation. It increases the appetite, diverts the mind, strengthens the voluntary muscles of defæcation, and stimulates the intestinal movements. When any of the voluntary muscles of defæcation are weak, considerable benefit can be gained by the performance of Swedish exercises every morning and evening. If constipation is due to want of activity of the intestinal musculature, the condition of the latter may be improved by deep abdominal massage. This should be directed particularly to the part of the colon in which the X-rays have shown that stasis occurs. Its efficacy is greatly increased if the first treatment is given during

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an X-ray examination, as the masseur can then see the exact position of the colon and can find what manipulations have most effect upon it. In cases of ptosis he can also learn how to manipulate the colon into its normal position.

Dyschezia is often associated with viscerop-tosis, both being due to weakness of the abdominal muscles. In such cases an efficient support is of great value. A. F. HURST.

CONSTIPATION, INFANTILE.—Constipation in infants may be either acute or chronic.

ACUTE CONSTIPATION

A more or less sudden stoppage of the action of the bowels frequently produces, as is well known, considerable constitutional disturbance from symptoms which are partly toxic and partly reflex in origin. More especially are the symptoms severe when the constipation has been preceded by loose or undigested stools. They may vary from the comparatively mild upset shown by a slight rise of temperature, with screaming, pain, disturbed sleep, and irritability, to an alarming disturbance with high fever and convulsions. **Treatment** consists in emptying the lower bowel by means of a small enema of soap and water or by a glycerin suppository, and the administration of a purge, castor oil ($\frac{1}{2}$ -1 dr.) being usually the most suitable.

CHRONIC CONSTIPATION

This complaint is frequently met with in babies who, as far as their nutrition is concerned, are thriving. Before dealing with such cases there are others to consider, and the less common cases will here be mentioned first.

1. **Rare causes of constipation.**—Congenital atresia of the bowel or anus and congenital dilatation of the colon (Hirschsprung's disease) require mention here. Any painful condition of the lower bowel will produce constipation, while there are, of course, many ailments of which this is a symptom. Mental deficiency in general, and cretinism in particular, must not be forgotten.

2. **Constipation with loss of weight.**—Setting aside partial starvation due to vomiting and constitutional diseases, this form of constipation is most commonly due to under-feeding. More especially is this common in breast-fed babies when the milk is gradually deteriorating in quality or quantity. Nor is it always easy to detect, for when the under-

feeding develops gradually the infant frequently accommodates itself to it uncomplainingly. This cause may be suspected when an otherwise healthy infant begins to grow thinner and to pass stools which are hard in consistence and small in bulk. The suspicion may be confirmed by noting the good effect of one or two full-sized artificial feeds given daily. It can be proved only by an analysis of the milk together with an accurate measurement of the infant's weight immediately before and after a feed from the breast.

Attention to the diet is here all that is necessary.

3. **Constipation in thriving babies.**—This is the commonest type of case which comes under treatment. The child is fat and strong, and not losing weight, but it suffers from colic and flatulence, which give rise to screaming and entail disturbed nights. The stools are hard, but large in bulk, and the bowels act only once in two or three days.

This type of case is seen chiefly among breast-fed babies, but is common in those fed on whole milk, whether citrated or desiccated. It also occurs in those fed on diluted milk. Its explanation would seem to be that the residue of the food in the intestine is too bulky and dry to be moved on at the normal pace by peristalsis.

Treatment of chronic constipation.—General measures must not be neglected. Of these may be mentioned fresh air, friction after bathing, and, in older infants, training the child to attempt a regular action of the bowels.

In very mild cases there are various additions possible to the diet which have a mildly aperient action: such are barley-water, cane-sugar, fruit juice, Mellin's food, or (in selected cases) cream or codliver-oil emulsion.

In severer cases, in which such mild measures as those are of no avail, we shall require the aid of drugs. Here there is so wide a field of choice that only those found the most satisfactory as routine measures will be mentioned. Grey powder (hydrargyrum cum creta, $\frac{1}{4}$, $\frac{1}{4}$, or $\frac{1}{2}$ gr.) should be used when the constipation is associated with the passage of faeces which are lumpy or undigested. It may be continued nightly for a week or so. If there are no signs of imperfect digestion, a routine treatment which suits most cases admirably consists of the following three measures, adjusted to the needs of the individual case.

(a) For the first few days, until the medicines

CONVULSIONS

are acting regularly, the bowels are opened by a small injection or suppository of soap or glycerin. By this means the use of purges in doses likely to produce pain is avoided.

(b) Liquid paraffin in sufficient doses to keep the stools from becoming hard should be given regularly. Generally between $\frac{1}{4}$ and 1 dr. should be given twice or three times daily; it is best administered separately from the feeds, unless the baby can be relied upon always to finish its bottles. The paraffin may be given pure, or in an emulsion, or with malt; and in selected cases may be usefully combined with codliver-oil emulsion, as can be done with the "Semplin" brand of paraffin. The doses given above are for the pure paraffin.

(c) Infusion of senna pods, made at home by soaking the pods in cold water for eight hours, should be given nightly. The usual dose for a baby is the infusion of from half to two pods, and this should be given quite regularly over a sufficiently long period to correct the constipation permanently. The home-made infusion is usually more certain in its results than the popular syrup of figs, though this latter is useful in emergencies.

REGINALD MILLER.

CONSUMPTION (*see* PULMONARY TUBERCULOSIS).

CONTRACTED GRANULAR KIDNEY (*see* under NEPHRITIS).

CONVULSIONS.—The occurrence of convulsions does not constitute a disease, but must be regarded as a symptom, the source of which requires investigation. A convulsion affords evidence of instability on the part of the brain or of its circulation. Whether this instability should be regarded as belonging to the category of epilepsy, or whether it may be ascribed to some adequate but temporary influence, is a question to which an answer is often difficult to find. A brief consideration of the general incidence of convulsions during infancy and adult life may be helpful in determining their cause in a particular case.

Infantile convulsions or infantile eclampsia.—The period of infancy is one during which convulsions are more easily provoked than at any other age, and provocation which is adequate in infancy becomes increasingly inadequate as years go by.

It is impossible to distinguish between many of the convulsions of infancy and those of true epilepsy, and it is especially difficult to

determine how far loss of consciousness is associated with the motor disturbance.

Infantile convulsions may result from antenatal disease or injury, as well as from accidents occurring at the time of birth.

Rickets predisposes the infant to convulsive attacks which are particularly liable to occur during the period of the first dentition.

A series of convulsions or a single attack may usher in any of the acute specific fevers and form part of the clinical picture of meningitis or encephalitis. It is more than probable that encephalitis in infancy is often labelled as "convulsions," the other results of the cerebral inflammation escaping recognition until the child has reached maturer years.

Convulsions in infancy are often laid to the account of intestinal worms, to gastro-enteritis, to phimosis, and to many other sources of peripheral irritation, but whether these would operate in the absence of some abnormal condition of the nervous system remains open to doubt.

Tetany is the most common spasmodic affection of early childhood to be mistaken for convulsions, but the characteristic attitude and complete retention of consciousness serve to distinguish it.

Gross organic disease of the brain in the form of tumour, abscess, or hydrocephalus may be responsible for convulsive attacks in children as in adults, although the diagnosis is often more difficult in the former.

Convulsions of adult life.—Apart from idiopathic epilepsy, convulsions in adults may be due to a number of different causes, of which the most important are poisoning by alcohol or lead, uræmia, cerebral syphilis or general paralysis of the insane, encephalitis, brain tumour and abscess, meningitis, eclampsia of pregnancy and the puerperium, arterio-sclerosis, aortic disease and heart-block, cerebral hæmorrhage and thrombosis, and any form of head injury. All these conditions must be regarded as exciting factors which in various ways play their part in producing convulsions. The influence they exert is in some cases intensified by an inherent predisposition or spasmophilia.

Lastly, some convulsions are undoubtedly of mental origin, various forms of hysterical fits belonging to this category.

E. FARQUHAR BUZZARD.

CONVULSIVE TIC (*see* TICS AND HABIT SPASMS).

CORNEA, AFFECTIONS OF

COPAIBA ERUPTIONS (*see* DRUG ERUPTIONS).

COPROLALIA (*see* TICS AND HABIT SPASMS).

CORNEA. AFFECTIONS OF.—Under this title are described—

1. ULCERATION.
2. INTERSTITIAL KERATITIS.
3. CORNEAL OPACITIES.
4. CORNEAL DEGENERATIONS.
5. CONICAL CORNEA.
6. NEURO-PARALYTIC KERATITIS.
7. LEAD DEPOSIT.
8. SYMPTOMATIC CONDITIONS.

Abrasions of and foreign bodies in the cornea are considered under EYE, INJURIES TO.

Affections of the cornea may be divided into ulcerations, interstitial inflammations, and new growths. The third group is rare, and need not detain us; the two former occur in both acute and chronic forms.

1. ULCERATION OF THE CORNEA

Purulent Keratitis, as this affection is also called, is always caused by the presence of an organism or its toxin, which attacks the corneal tissue after preliminary destruction of some part of the epithelium, brought about by lowered resistance or some trivial injury.

Some ulcers, having once started in this manner by destruction of the first line of defence, spread no farther, and under appropriate treatment at once begin to heal; whilst others continue to extend either superficially or into the deeper layers, and eventually lead to perforation. The course which an ulcer pursues depends on the virulence of the organism or toxin, or on the resistance offered by the second line of defence, the antitoxic properties of the blood in the underlying tissues.

From a general practitioner's standpoint, the question to determine is not so much the kind of ulcer as whether ulceration actually exists, a matter about which he is frequently in doubt. The presence or absence of an active ulceration, however, can be decided in a moment by the application of a drop of fluorescein, as described under EYE, EXAMINATION OF, and, by employing the other two methods of examination set out in that article, the nature of any inflammation, without ulceration, can then be determined with more or less accuracy. In some cases the ulcer can be seen with the naked eye. If there is any difficulty in separating the lids, owing to the photophobia, retractors must be used, as all rough handling

with the fingers is dangerous. Whenever photophobia or a pricking sensation is complained of, it may be taken for granted that corneal inflammation exists, and the diagnosis can be confirmed from the signs and symptoms about to be enumerated.

The signs and symptoms common to all inflammation of the cornea are: (1) *Ciliary injection* (*see* EYE, EXAMINATION OF). (2) *Loss of brilliancy* in the corneal reflex. (3) *Pain*, which varies from a mere pricking sensation to an intense irritation that may lead to severe photophobia, the eyes being kept tightly closed to exclude all entry of light. The pain may pass off after the first day or two from the beginning of the inflammatory reaction—a bad sign, as will be explained later. (4) *A copious discharge of water* from the eyes. The discharge is not mucoid, and therefore does not give rise to any sticking of the lids after sleep, a point which serves to distinguish corneal affections from simple conjunctivitis.

The prognosis in any given case varies with the nature and extent of the inflammation, or the complications likely to ensue; these will be considered under each separate variety of ulcer.

Treatment.—Unless there are special indications to the contrary, of which there are very few, the fundamental treatment of all corneal conditions, whether ulcers in any position or interstitial inflammation, is the same. The conjunctival sac must be kept clean and, as far as possible, aseptic by washing out the eye several times a day with lotion, perhaps the best in these cases being boric acid (10 gr. to the ounce), and atropine (1 or $\frac{1}{2}$ per cent.) in the form of ointment or drops must be applied inside the eye two or three times a day to dilate the iris, and keep it dilated, until the eye is perfectly well. A common notion prevails that atropine should not be used in cases of corneal ulcer situated near the sclero-corneal margin, because in the event of perforation prolapse of the iris is more likely to occur when the pupil is fully dilated; this belief has no foundation. It is doubtful if prolapse of the iris can ever be prevented after perforation, whatever position the iris occupies at the moment of evacuation of the anterior chamber, except when the ulcer is small and situated exactly in the centre of the cornea, and the pupil is fully dilated. Compared with the importance of using atropine, this point is not worth consideration.

An eye with inflammation of the cornea must

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be shaded, and in some cases with a clean conjunctival sac more rapid healing is promoted if the eye is covered with a bandage. When, however, conjunctivitis is also present, bandaging is, in my opinion, positively harmful, whereas the use of a shade or neutral-tinted glasses to exclude light, and thus rest the eye, is never wrong. In addition to these methods of treatment common to all corneal inflammations, special means must be adopted in certain cases, without which an ulcer may become very chronic, and not only fail to heal but lead to complications that may be serious. They will be mentioned under the headings dealing with the different varieties.

The commoner of these varieties are five in number: (a) Epithelial, (b) Phlyctenular, (c) Hypopyon, (d) Catarrhal, (e) Ulcers secondary to purulent conjunctivitis, e.g. ophthalmia neonatorum. Each must be carefully distinguished and recognized if treatment is to be successful.

(a) EPIITHELIAL ULCERS.—As their name implies, these ulcers are confined to the epithelium. Analogous to vesicles formed on the skin in herpes, they are always extremely painful, causing great irritation, with a pricking sensation, lachrymation, and blepharospasm, for the nerve-endings of the cornea are merely exposed and not destroyed. According to their size, distribution, and extent they have received special names, but the treatment is practically the same in each case. If the destruction of the epithelium occurs in many spots, isolated and scattered all over the cornea, the ulceration is called *superficial punctate keratitis*; if the ulcers are larger and tend to radiate out in lines from one central spot, they are called *stellate*; whereas if they extend in branches all over the surfaces of the cornea like the branches of a tree they are described as *dendritic*. Such ulcers are often suspected from the symptoms described above, and can readily be demonstrated by the instillation of a drop of fluorescein. Unless definitely looked for in this way they may escape observation, since the inflammation, being confined to the epithelium, shows no hazy area visible to the naked eye as when Bowman's membrane and the substantia propria of the cornea are invaded.

Superficial punctate keratitis, although it heals rapidly, is very liable to recur if untreated; it is common for a patient to complain of a bloodshot eye accompanied by intense prick-

ing pain and lachrymation, which pass off in a few hours, only to be followed a day or two later by a repetition of the symptoms—a state of things which extends over a period of some weeks. These ulcers occur after febrile attacks, never perforate, but tend to spread all over the cornea superficially, and may, if not recognized, extend through Bowman's membrane into the deeper layers, and thus after healing form nebulæ. In addition to the use of atropine, the eye should be washed out with boric-acid lotion containing some weak sulphate of zinc, since the condition is almost always associated with chronic conjunctivitis. Occasional painting of the lids (once or twice a week) with the weaker silver preparations is often of great help in rendering them more healthy and thus influencing the healing of the ulcers.

Stellate and dendritic ulcers are treated in the same way as superficial punctate keratitis, but it is nearly always necessary to apply some form of caustic to the ulcer itself in order to arrest its progress, since there is not the same tendency to rapid healing. An effectual plan is to paint the ulcer with pure carbolic acid, which is a local anæsthetic as well as a caustic. The cornea should be cocaineized thoroughly, and 2-per-cent. fluorescein instilled in order to map out the whole extent of the ulcer; then, after drying the cornea carefully with a clean piece of blotting-paper, apply the head of a wooden match, sharpened to a round point, and dipped in pure carbolic acid, to the ulcer over the parts that stain. It will be noticed that when the carbolic touches the cornea the tissue becomes white, showing that superficial destruction is complete. After the surface has been dried again with blotting-paper the eye is bandaged for twenty-four hours. The process may have to be repeated several times at intervals of two or three days until all trace of ulceration has disappeared as shown by the fluorescein stain.

Ulcers of this variety are often found in cases which otherwise only appear to be ordinary but troublesome conjunctivitis, and are very easily overlooked. They are liable to recur; and one of the best ways of avoiding this is to wash the eyes occasionally for a few weeks with some lotion containing zinc, and to instil now and then a drop of dionine (5 per cent.), preferably in the morning.

(b) PHLYCTENULAR ULCERS.—These are most commonly seen in children, and are rare in adults. They give rise to characteristic symptoms, with which all practitioners are familiar.

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The subjective symptoms are sometimes much exaggerated, and the photophobia often reaches so extreme a degree that the lids are kept firmly closed in all circumstances and it is practically impossible to separate them sufficiently to get a satisfactory view of the cornea without the use of retractors. The edges of the lids and the skin over them become excoriated and raw owing to the constant moistening by secretion, and this leads to further aggravation of the blepharospasm. It is not altogether a bad sign, as it shows that the ulcer is more or less superficial and is not extending rapidly into the deeper parts. There is also ciliary injection, with other signs of corneal ulceration, but the obstinate closure of the lids often persists long after the ulcer has healed. This is largely a neurotic condition, but its continuance has a depressing effect both on the parents and on the child, causing them to believe that the ulcer is still present in an acute form; it provokes much needless anxiety and disappointment. One of the best ways of treating it is to put the child's head and face into a basin of cold water, holding it there for an appreciable time. In some cases slitting of the external canthus has to be resorted to.

Phlyctenular ulcers may occur on the cornea in the first instance, or may result from direct extension from conjunctival phlyctenules. In whichever way they start, they run the same course and tend to spread over the surface as well as into the deeper layers. If the resisting power of the tissue is lowered the ulcer is liable to end in perforation, though this is not the usual termination. The dangers of perforation must be thoroughly understood by the practitioner in order that he may be on the alert and use all the means in his power to avoid it. When an ulcer perforates, the first effect is that the aqueous is discharged, and during its escape the iris is washed out through the opening of the cornea and thus becomes entangled in the lips of the wound. Owing to the inflammatory processes which have been going on, the iris readily adheres, becomes fixed in this new position, and if left too long cannot be disengaged. At the same time it forms an efficient plug, closing up the hole in the cornea, and giving an opportunity for the anterior chamber to re-form, which it does in a few hours. This plug, however, is not strong enough to withstand the normal intra-ocular pressure, and, consisting partly of iritic and partly of corneal tissue, gradually bulges and

produces an anterior *staphyloma*. Moreover, an eye in which the iris is adherent to any part of the back of the cornea (*anterior synechia*) may become the subject of secondary glaucoma. When the anterior chamber is evacuated through a central hole in the cornea, the anterior capsule of the lens comes into contact with the cornea and remains so for an appreciable time; the inflammatory process in the cornea is communicated to the lens capsule, and a fibrous deposit occurs at this spot exactly in the middle line (*anterior polar cataract*). This will interfere with vision after healing of the ulcer and after the anterior chamber has re-formed.

Ulcers which extend superficially form large or small infiltrated areas consisting of blood-vessels and granulation tissue. The infiltration first extends between Bowman's membrane and the epithelium (*phlyctenular pannus*), wherever the latter happens to be intact, and later penetrates to the substantia propria of the cornea.

Another form which phlyctenular ulcers may take is that known as *fascicular*, in which the ulcer spreads from the limbus in the shape of a narrow cone with its apex directed towards the centre of the cornea, and eventually, if not treated promptly, stretches right across to the opposite side. A leash of superficial blood-vessels follows this cone-shaped area, and from this the name is derived. It is a particularly stubborn variety of phlyctenular ulcer, and the special line of treatment required is to cauterize the advancing apex with the actual cautery; personally, I find that healing is accelerated by cutting off at the same time the leash of vessels at its origin near the limbus.

In all cases of phlyctenular ulcer the general health must carefully be attended to by means of tonics, good food, and change of air, preferably at the seaside. It is astonishing how quickly the eyes recover in such circumstances, but, as the patients have to return sooner or later to unhealthy surroundings, the ulcers are very liable to recur.

Bearing in mind the dangers of perforation, atropine must be pushed until full dilatation of the pupil is procured, and maintained throughout the whole course of the corneal ulceration; no supposed theories of the greater liability to anterior synechia with a dilated pupil should deter one from pressing it to its full effect:

(c) **HYPOPYON ULCER.**—This form of acute ulceration of the cornea, known also as *Ulcer*

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Serpens, is especially virulent, and extends very rapidly, causing perforation in a few days, with inclusion of the iris, perhaps followed by sup-puration in the globe, and loss of the eye in a comparatively short time. It generally starts with some abrasion caused by an infected foreign body. In the very early stages it is somewhat painful; but the pain soon passes off owing to rapid destruction of the corneal nerve-endings, and lulls the patient into a state of false security, so that advice is not sought until the hypopyon has extended sufficiently to interfere with the sight. The pus in the anterior chamber is caused by the passage of toxins formed by the organisms (pneumococci) on the surface through the cornea into the aqueous humour, where they act on the iris and produce an intense iritis. This leads to an exudation of leucocytes, etc., from the vessels of the iris, and the pus thus formed sinks down to the bottom of the anterior chamber. Hypopyon has been mistaken for an infiltration in the lower part of the corneal tissue, but the dense white substance with a perfectly straight well-defined horizontal border, corresponding to the upper level of the fluid, should always excite suspicion, and the presence of a sloughy-looking ulcer should confirm the diagnosis. A small hypopyon, however, often requires to be looked for carefully. That the pus is sterile, and must not be treated in the same way as pus in other parts of the body, cannot be too often insisted upon. There is no need to treat the hypopyon apart from the ulcer, but if it is found necessary to open the eye, a procedure which may become imperative should increased tension occur, an attempt may sometimes be made to remove the pus, since it is then liable to infection from the exterior. More often, if proper treatment is directed to the ulcer, the hypopyon clears up spontaneously.

Hypopyon ulcer is not infrequently accompanied by increased tension, which further complicates an already very dangerous condition, and calls for urgent treatment. The secondary glaucoma is produced by the exudate acting as a means of uniting the iris to the back of the cornea and thus blocking the angle of the anterior chamber. This causes a return of pain which has been in abeyance.

Treatment.—In the early stage the ulcer, which is generally situated about the centre of the cornea, should be cauterized either with pure carbolic or with the actual cautery; I prefer the latter method in any but very mild

cases. The cauterization must be applied freely and extend as deeply into the corneal tissue as is safe without causing perforation, and also laterally for a short distance into normal cornea. The resulting nebula, which often clears up to a considerable extent, may be disregarded, since the problem is one of saving the eye with a certain amount of useful sight, and not of restoring perfect vision, which is practically impossible.

If the ulcer shows no sign of clearing up after this treatment, it may be necessary to open the anterior chamber by making an incision through the base of the ulcer; especially is this imperative if the tension becomes raised. Later, the adherent iris which inevitably results must be dealt with in order to avoid secondary glaucoma, and in some cases it may be advisable to perform an iridectomy at the same time that the eye is opened.

In order to inhibit the growth of the pneumococci on the ulcerated surface, various lotions are employed, the most usual being perchloride of mercury (1 in 7,000), hydrogen peroxide, and quinine hydrochlorate (1 per cent.). The conjunctival sac must be washed out as often as in a case of ophthalmia neonatorum (*see* CONJUNCTIVITIS).

The presence of pus in the anterior chamber seems to suggest to some practitioners the use of fomentations, but it must be remembered that the pus is sterile and is due to toxins produced by the organism on the surface, and that these organisms will grow more vigorously under the stimulus of heat.

If the eye be kept open, the conjunctival sac washed out as often as is practicable, and the ulcer treated by the special means advocated, many of these cases will get well though with a certain amount of damage to the sight.

(d) CATARRHAL ULCER.—This ulcer is so called because it is due to catarrh of the lids; it occurs in the form of small round or oval ulcers situated just inside the limbus at various sites round the circumference. The characteristic feature is that there is always a small area of normal cornea between the ulcer and the sclero-corneal margin. Several such ulcers often exist together and, if not treated, or if treated improperly, coalesce to form one large crescentic marginal ulcer. Perforation only occurs very occasionally. In addition to the use of atropine, measures must be taken to decrease the conjunctivitis. Perhaps the best lotion for this purpose is a weak solution of sulphate of zinc. Painting the lids with prot-

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argol (20 per cent.) or silver nitrate (1 per cent.) once or twice a week for the first week or so, according to the severity of the conjunctivitis, often hastens recovery.

(e) Ulceration secondary to conjunctivitis is considered under CONJUNCTIVITIS.

2. INTERSTITIAL KERATITIS

In Parenchymatous Keratitis, as this condition is also called, deep infiltration occurs without destruction of the surface. There are three varieties—(a) syphilitic, in which the syphilis is practically always inherited, (b) keratitis profunda, and (c) sclerosing keratitis. In all alike the cornea in the affected area has a ground-glass appearance, due partly to the actual infiltration and partly to oedema of the cornea.

(a) In **SYPHILITIC KERATITIS** the infiltration, accompanied by an intense and deep vascularity, starts at the limbus in the upper and lower quadrants, and simultaneously and rapidly covers the whole of the cornea, giving rise to a pinkish-white opacity, which effectually obstructs all rays of light and renders the patient for all practical purposes blind. The appearance produced is known as a "salmon patch." However severe the inflammation, there is no ulceration. Sooner or later both eyes are affected, though for the first few weeks the inflammatory process may be confined to one eye only.

The commonest age for syphilitic keratitis to occur is between 5 and 15 years.

(b) In **KERATITIS PROFUNDA** the actual infiltration has the same clinical appearance, though it is never so acute nor attended with the formation of so many new vessels. Sometimes the blood-vessels are absent, while in other cases there are at the most but one or two long unbranching vessels, which can only be made out with difficulty. Moreover, the inflammatory process is more likely to be confined to one eye than in the syphilitic variety, the other being seldom affected.

(c) In **SCLEROSING KERATITIS** the inflammation spreads in patches directly from the sclera to the cornea. The patches are of irregular shape, are much slower in extending than in keratitis profunda, and appear in many different places round the limbus. They do not cover the whole cornea as in the syphilitic variety, but often involve the deeper structures (e.g. the ciliary body) before they have advanced very far. Both keratitis profunda and sclerosing keratitis are almost always due to toxic infection from a septic focus somewhere in the

body, e.g. teeth, nose, intestinal tract. A thorough investigation should therefore be made in order to detect the source, if possible, so that appropriate general treatment can be carried out; otherwise the condition tends to become very chronic, the absorption of the infiltration taking place very slowly and often being incomplete.

In interstitial keratitis there is a rounded exudation into the deeper layers of the cornea together with a formation of new vessels from the deep circulation surrounding the limbus. Since this part of the cornea is developmentally part of the uveal tract, the complications to be expected are iritis, cyclitis, and choroiditis.

The **diagnosis** of interstitial keratitis is not difficult if the following points be borne in mind, viz. the ground-glass appearance of the cornea, ciliary injection, some photophobia (which varies greatly and may be absent altogether, as in some cases of keratitis profunda), and the negative effect of fluorescein-staining.

The **prognosis** is generally favourable so long as no complications arise; but should these occur, the sight of the eye may be very seriously affected or lost. It is well known that some of the worst cases of syphilitic interstitial keratitis clear up so completely that the vision, after having been apparently hopelessly lost, recovers to $\frac{1}{2}$. The knowledge that this termination is a possible one constitutes a valuable asset at the time when the appearance of the eye and the effect on the sight are so alarming to all concerned that a pessimistic view of the ultimate result seems to be quite legitimate.

A certain amount of opacity, however, is almost sure to be left, causing defective sight, and for this the patient should, as a rule, be prepared.

Treatment consists in hot bathing of the outside of the eye with the lids closed, instillation of atropine (1 per cent.), general treatment, and dark glasses to protect the eyes from light.

Antisyphilitic remedies would appear to be the ideal form of general treatment in the syphilitic cases, but the result has been disappointing hitherto, though they should certainly be tried.

3. CORNEAL OPACITIES

All inflammations of the cornea which extend deeper than Bowman's membrane are

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liable to leave scars when the healing process is complete, because of fibrous-tissue changes in the substantia propria of the cornea. The affected area seldom becomes entirely transparent again, though the amount of absorption of the opaque tissue is often surprising; the younger the patient, the better the chance of its clearing. *Nebula* is the term applied to slight opacities, *macula* indicates one of moderate degree, while by *leucoma* is meant an opacity of great density, and therefore white, usually the result of hypopyon ulcer. These scars interfere considerably with vision, partly because of the density of the opacity, partly from the irregular astigmatism they give rise to. As the original inflammation dates so far back that the patient may have forgotten all about it, *nebulæ* should always be looked for in any case in which defective vision cannot be explained in other ways. Very faint *nebulæ* are often difficult to see, but they can generally be recognized by oblique illumination if the patient be directed to turn the eye upwards.

Treatment. — Many attempts, in various ways, have been made to promote absorption of the fibrous tissue in corneal scars, but so far no specific has been discovered. Yellow-oxide-of-mercury ointment is indicated. Subconjunctival injections have been given with benefit in some cases.

4. CORNEAL DEGENERATIONS

Degenerations of the cornea give rise to opacities which, if they occur in old *nebulæ*, are seen as discrete glistening white hyalin deposits. Their only significance is that they indicate the improbability of any further absorption. Another form of degeneration (fatty) is *arcus senilis*, situated in the upper and lower quadrants of the cornea just inside the limbus, often joining to make a complete circle. It never spreads to the centre.

5. CONICAL CORNEA

This rare condition only occurs in adult life and often gives rise to intense headache after reading and great deterioration of vision owing to the high astigmatism which results. The treatment is not very satisfactory; many operations have been devised, but even in the hands of experts they do not always produce the desired results, and they should never be undertaken by the general practitioner. Sight may be considerably improved by the wearing of very high cylinders, though the glasses are clumsy and heavy for the wearer.

6. NEURO-PARALYTIC KERATITIS

Keratitis of this variety is due partly to trophic changes induced by paralysis of the fifth nerve and partly to degeneration of the epithelium. There is generally no pain, the cornea is hazy all over, and often ulcerated, and shows no tendency to heal. The best treatment is to suture the lids together after carefully paring the edges for one-third or half their length.

Closely allied to this condition is the *Keratomalacia* seen in marasmic children.

7. LEAD DEPOSIT IN THE CORNEA

Shows as a dense white opacity in some part of the cornea and looks something like a degeneration, but differs in having a sharply-defined margin separating it from the rest of the cornea, which is perfectly healthy and shows normal brilliancy. Degeneration in an old *nebula* never has so discrete an outline, for the inflammatory process from which it originated always extends slightly into the surrounding zone and makes its outline uneven. Lead deposit is not now so common as when lead lotions were used freely for any inflammation of the eye. Such lotions should never be ordered in any circumstances. If a deposit is found, it must be scraped off deliberately and methodically under cocaine.

8. SYMPTOMATIC CONDITIONS OF THE CORNEA

Certain conditions of the cornea are merely symptomatic; such are Striate Keratitis, Bullous Keratitis, and Keratitis Punctata.

Striate Keratitis is seen on magnification as opaque grey lines often placed vertically, but sometimes forming a sort of network and running in all directions over the cornea; they are due to swelling and folding of the posterior layers of the cornea, which are swollen as the result of inflammatory infiltration.

Bullous Keratitis is found in cases of increased tension of long standing, and is therefore common in old glaucomatous eyes.

Keratitis Punctata is the only external sign of irido-cyclitis, apart from the signs and symptoms common to this and to iritis, and is described under UVEAL TRACT, AFFECTIONS OF. It often has to be examined for with great care, as it is easily overlooked, and it is only in the obviously severe cases that the spots can be seen with the naked eye.

MALCOLM L. HEPBURN.

CORNS

CORNS.—A corn (Lat. *clavus*) is a localized hypertrophy of the corneous layers of the epidermis.

Etiology.—The condition is perhaps the simplest illustration in human pathology of the law of growth reaction to a chronic stimulus, for wherever a corn is found antecedent pressure or friction may be surmised. By far the most common situation, therefore, is on the dorso-lateral aspect of the little toe, where both the pressure and friction of tight and ill-fitting shoes are naturally concentrated. Congenital and traumatic malformations are often responsible, and flat-foot invariably produces corns at definite points of pressure on the plantar aspect. Various occupations, and certain sports, such as rowing and golf, tend to produce a somewhat similar condition on the palmar aspects of the hands and fingers. These "callosities," as they are called, are protective, and of no pathological interest.

Histopathology.—The microscopic picture is one of simple hypertrophy and hyperplasia of the corneous layer. Towards the centre of a corn the horny substance, owing probably to faulty keratinization or disintegration, may undergo softening, and can sometimes be withdrawn as the erroneously termed "core" or "root." Such an operation will obviously not cure the condition. Prevented from upward growth by the very cause which produced it (i.e. the boot or shoe), the apex of the cone-shaped clavus grows downwards into the soft tissues. Inflammatory reaction is a painful and common sequel, and is Nature's attempt to rid itself of a foreign body, an attempt which is occasionally successful if acute suppuration occurs.

Symptomatology.—The symptoms and signs are too well known to need a lengthy description. The appearance of a corn necessarily varies with its situation, and when there is pressure and friction between the toes, a so-called soft corn is the result. This is invariably associated with hyperhidrosis, and septic complications are common.

Diagnosis.—A corn on the plantar aspect of the foot, particularly if bilateral, and painlessly discharging a serous fluid, should at once arouse a suspicion of *tubercle dorsalis* as the underlying factor. I have seen two such cases "missed," and fruitlessly treated for weeks.

Treatment.—The cure of a corn is intimately connected with the removal of its cause. High-heeled, pointed shoes, which act as an inclined

CORONARY ARTERIES, DISEASES OF

plane and cram the toes chaotically on each other, must be replaced by others of a more serviceable pattern, and the corn protected from pressure by a small ring of adhesive felt. When inflammatory manifestations have subsided the horny overgrowth should be softened with repeated applications of a 20-per-cent. salicylic-acid plaster or 10 per cent. salicylic acid in flexile collodion. The plaster is much more rapid in action, and after some forty-eight hours' application the foot is thoroughly soaked in hot water. The corn can then usually be gently levered from its funnel-shaped bed. Soft corns should first be rendered aseptic with perchloride fomentations or calomel powder, and then carefully destroyed with a caustic, such as trichloroacetic acid applied on a pointed match. Recurrences can only be avoided by strict attention to the footwear.

HENRY SEMON.

CORONARY ARTERIES, DISEASES OF.

—The importance of the coronary arteries in disease lies almost solely in the fact that they convey nutriment to a vital tissue, the myocardium. Injury to these channels, therefore, expresses itself in interference with the structure and functions of the cardiac muscle. When the coronary disease is diffuse, as it is generally in the commonest form of case, that of chronic atheroma, the resulting fibrosis and dystrophy of the myocardium are also diffuse, and the functions of that tissue are affected throughout. Clinically, therefore, the picture is that of chronic myocardial degeneration (*see MYOCARDIAL DEGENERATION, PROGRESSIVE*).

Sometimes, however, there is gross disease of a main coronary trunk or a large branch. This, together with its results, is so different, pathologically and clinically, that it must be described separately.

Coronary thrombosis usually occurs in an area where the channel is already narrowed by chronic disease, especially atheroma and syphilitic arteritis. Possibly the occluding clot is sometimes laid down in instalments, but even so it is the last instalment that is responsible for most, if not all, of the clinical phenomena that may manifest themselves. For all practical purposes, therefore, coronary thrombosis may be looked upon as of sudden occurrence. **Coronary embolism** is a rare complication of ulcerative endocarditis and of chronic valvular disease. When a large branch or a main trunk is suddenly plugged in this way the sequence of pathological and clinical

events is identical with that which follows on thrombotic closure of a similar vessel, with this exception, that infective embolism may be followed by the formation of a coronary aneurysm (*see below*).

The *pathological changes* that follow sudden occlusion of a large coronary artery are (1) necrosis of the area of myocardium thus deprived of its blood supply, the dying tissue becoming surrounded and marked off from the living by a zone of inflammatory reaction (anæmic infarction); (2) absorption of the dead muscle, which is replaced by cicatricial fibrous tissue. If the patient survive long enough, and yet the area affected be large, this scar may be bulged outwards by the intracardiac pressure, to form a cardiac aneurysm, which may rupture into the pericardial sac. In any case the pericardium covering the damaged cardiac muscle is inflamed. Localized disease of this kind is nearly always situated in the wall of the left ventricle near the apex.

Symptomatology.—(1) Death may ensue immediately, leaving no time for symptoms. (2) In a patient known to have serious heart disease, the occurrence of a severe and prolonged attack of angina pectoris, accompanied and followed by intense dyspnoea, rapid pulse, vomiting, and enfeeblement of the cardiac sounds, may suggest a diagnosis of sudden coronary occlusion. If a pericardial rub also develops, the diagnosis is further supported. The patient may succumb to such an attack after a struggle for thirty-six or forty-eight hours. (3) Aneurysm of the heart scarcely ever reaches such a size as to give rise to distinctive physical signs, but the presence of an abnormal area of pulsation in the neighbourhood of the cardiac apex has in one or two instances permitted of a successful diagnosis. (4) Sudden rupture of the heart, whether preceded or not by the development of an aneurysm of the heart, is quickly fatal; if the interval between rupture and death be sufficiently long for symptoms to develop, the picture is that of sudden cardiac collapse, sometimes associated with pericardial friction or rapid increase in the area of cardiac dullness.

The **prognosis** is always bad. Sudden coronary obstruction only occurs in hearts that are profoundly diseased. To a burden that is almost intolerable this further disablement is suddenly added, and the majority of patients succumb within a day or two at most.

Treatment.—The immediate indications are

absolute rest in whatever position the patient prefers, and inhalation of amyl nitrite. This will give relief from pain, and alleviation of the heart's burden of work. If it fails, morphia is necessary. If these measures are successful in piloting the patient through the period of immediate peril, he must still be kept quite quiet, and nitrite of soda or one of the other nitrite compounds with an action less evanescent than that of amyl nitrite should be given continuously. If there is reason to suppose that syphilis is responsible for the coronary disease, treatment by mercury and iodides should be begun as soon as the patient seems fit to stand it.

Coronary aneurysm.—As these aneurysms never attain to a size larger than an ordinary horse-chestnut, their sole clinical importance lies in the possibility of sudden death from intrapericardial rupture which they introduce, usually into a case of ulcerative endocarditis.

CAREY COOMBS.

CORPORA QUADRAGEMINA, LESIONS OF (*see NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF*).

CORPULENCE (*see OBESITY*).

CORPUS CALLOSUM, LESIONS OF (*see NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF*).

CORYZA (*syn.* Common Cold, Acute Catarrhal Fever).—Coryza is the term applied to acute or chronic catarrh of the upper air-passages, including the pharynx.

Etiology.—No age or class is immune from attacks which, though commonest during the cold or winter months, are far from exceptional during the spring and early summer. The attacks may occur in epidemics. In some persons nasal catarrh is practically constant.

Any irritant to the nasal mucous membrane is liable to produce a reaction, which varies in duration according to the nature of the irritant. If the irritant is non-infective, the attack may only last a few minutes or hours, but if infective, all the changes which accompany infection elsewhere will develop, and may either be followed by cure or become chronic. Dust, pollen, or other nasal irritants of less definite origin may start the process and render the nasal mucous membrane suitable for the implantation of infections. A lowered general vitality also increases the tendency to coryza. Local abnormalities have a similar

CORYZA

effect. The nose may be too narrow or too patent, the latter being due sometimes to too vigorous surgical interference. Similarly, the presence of polypi or adenoids and large tonsils is detrimental, inasmuch as they obstruct the air entry or provide a focus for the spread of infection. A pneumococcus is nearly always present at the end of an attack, and also in chronic cases.

Symptomatology.—The attack may begin with sore throat or with malaise, shivering, slight rise of temperature, and other febrile symptoms. Very commonly the onset is preceded by an increased feeling of well-being, the air-passages being exceptionally free. The early stages are characterized by fits of sneezing, followed by a free liquid nasal secretion. The conjunctivæ are injected, the Eustachian orifices are obstructed, the teeth may ache, and various rashes may develop. The nasal passages are gradually blocked, leading to mouth-breathing, while the discharge becomes thicker and is removed less easily. In the course of two or three days the obstruction becomes intermittent and the discharge more easily got rid of; finally a return to the normal takes place. During this period various uncomfortable sensations or pains may develop in different parts of the head, due to involvement of the nasal sinuses. Not infrequently the catarrhal process spreads to the larynx and bronchi, thus producing the symptoms of a mild laryngitis or bronchitis.

Diagnosis.—There is little possibility of confusion. Hay fever should not be separated pathologically from this condition. In chronic or recurrent affections, one of the sinuses may be persistently affected, giving rise to recurring attacks and general ill-health in the interim.

Prognosis.—Acute coryza generally runs its course in ten days, but often the attacks are much shorter. The chronic variety is persistent during the winter, and in some cases through the summer also. When colds recur frequently there is considerable liability to acute or chronic inflammation of the sinuses.

Treatment.—This is of little avail. Most people take little notice of the acute condition beyond refraining from going out in the evening during the attack. Subjects in whom it is liable to spread to the bronchial system are well advised to remain in the house till the attack has cleared up. Such patients should take a sharp purge and, before going to bed, a hot bath followed by a hot drink of lemonade or whisky and water, and 15 gr. of Dover's

COUGH

powder. This will produce a considerable amount of comfort. If headache is severe it may be assuaged by aspirin, phenacetin, or some similar preparation. Local applications of heat, as by a sponge wrung out of hot water, are of assistance. When the nasal discharge is viscid and difficult to remove, relief may be obtained by the employment of a nasal douche, consisting of an eggspoonful of salt dissolved in a tumblerful of water, or some more elaborate nasal douche. A tonic at the end of the illness will frequently assist convalescence. Those who are liable to frequent attacks may try the effect of a nasal douche containing an antiseptic such as permanganate of potash, night and morning, as a routine measure. Anti-catarrhal vaccines injected every third, fourth, or fifth week throughout the winter tend to prevent recurrences. Special vaccines directed against hay fever are sometimes successful (see HAY FEVER). It is of the greatest importance to ascertain that the nasal mucous membrane and pharynx are not abnormal, and that no focus of chronic infection exists in the upper air-passages.

CHARLTON BRISCOE.

COUGH.—Coughing is almost always reflex in origin, and results from irritation of some part of the sensory branches of the vagus. A possible exception to this is the cough of *hysteria*, which may be voluntary, but even in this instance some slight reflex irritation is probably associated.

The commonest site of stimulation is the *air-passages*, from the vocal cords to the finest bronchi. It is therefore an almost constant accompaniment of laryngitis in all its forms, laryngeal ulceration and neoplasms, tracheitis, bronchitis, bronchiectasis, broncho-pneumonia, and foreign bodies in the air-passages. Pressure upon the air-passages, as by an aneurysm, goitre, mediastinal tumours or glands, also excites coughing, partly by direct stimulation and partly by collection of mucus in their lumina. All forms of *pulmonary disease*, acute or chronic, are accompanied by cough, and whether this depends upon irritation of the smaller bronchi is a matter of no practical importance. Irritation of the *pleura*, as in pleurisy and pleural effusions, is also responsible, but to a less degree. Disease of the *heart and pericardium* is another cause arising from intrathoracic viscera. Irritation of the *recurrent laryngeal nerves* by mediastinitis, mediastinal tumours, glands, or aneurysm of the aorta or great vessels, may cause cough by

COUGH

COXA VALGA

spasm of the vocal cords. Among the commonest exciters of the reflex are abnormal conditions of the *pharynx*, *naso-pharynx*, and *nose*, such as adenoids, enlarged tonsils, a long uvula, various forms of pharyngitis, rhinitis, and nasal polypi. The "*stomach*" cough common in dyspepsia, especially in children, is generally due to irritating eructations, but may possibly arise by the transmission of impulses from the stomach or from the diaphragm. *Intestinal* cough is said to occur as the result of digestive disturbances, especially in children, in whom worms are regarded as an exciting cause. An *aural* cough from irritation of the external auditory meatus, as by wax, is well recognized, and is due to stimulation of Arnold's nerve.

The **character** of the cough may be of considerable help in the diagnosis. The following varieties are fairly distinctive:—

1. The *hacking* cough is short, dry, irritable, and frequently repeated. It may be due to slight catarrhal conditions of the pharynx or upper air-passages, and is characteristic of early phthisis. In the latter it is often most noticeable in the evening or early morning.

2. *Paroxysmal* cough is marked in whooping-cough, when it is followed by the whoop, vomiting, and the bringing up of much viscid sputum. A similar cough occurs with caseous intrathoracic glands in children, but without the whoop. It is paroxysmal also with mediastinal tumours, advanced phthisis, gangrene of the lung, and pleural effusion. In bronchiectasis it occurs chiefly in the morning, and results in profuse expectoration of foul sputum. In the case of children, if its incidence is nightly, it generally denotes a long uvula or enlarged tonsils or adenoids.

3. A *harsh, hoarse "croupy"* cough accompanies affections of the larynx. If there be destructive ulceration of the vocal cords, however, it may become toneless and "*whispering*" in character. In the case of a foreign body in the larynx or laryngeal papilloma the cough is often "*squeegie*" like.

4. The names "*brassy*," "*chicken*," "*cow*," and "*gander*" cough have all been given to the form which is heard in laryngeal paralysis due to affection of the recurrent laryngeal nerve. It is associated most commonly with aneurysm of the aortic arch and mediastinal tumours.

5. The "*barking*" cough occurs in hysteria. It is loud and explosive, apparently produced

without effort, and unaccompanied by expectoration.

In pleurisy and pneumonia the cough is *short, restrained, and painful*.

Coughing may be slight and ineffectual if there is much prostration, as in the later stages of phthisis or in conditions of grave toxæmia, and its cessation may under such conditions be of serious import. It is also feeble in paralysis of the larynx or muscles of respiration and in states of abdominal distension.

The **treatment** of cough is that of the condition which causes it and will be found under the proper headings.

FREDERICK LANGMEAD.

COXA VALGA.—A deformity of the femur in which the angle between the neck and shaft is increased. It may be *congenital*, with or without dislocation of the hip-joint, or *acquired*, as a result of loss of function of the limb, from paralysis, disease, or injury. After amputation of the thigh, coxa valga usually develops in the stump, and to some extent compensates the adduction of the hip-joint that is usually present. Traumatic coxa valga may be the result of fracture in the region of the trochanters, following treatment by extension in the abducted position; here it compensates any shortening that may result from the fracture. Sometimes coxa valga results from rickets, if the child is kept off its legs for a long time. A static or functional variety has also been described.

Symptoms and treatment.—There is a slight limp, and the hip movements are altered; abduction and external rotation are increased, adduction is diminished. The great trochanter is less prominent than normal, and the limb may be lengthened, producing secondary lumbar scoliosis. Treatment is usually unnecessary, but osteotomy of the femur for this condition has been described.

C. W. GORDON BRYAN.

COXA VARA.—A deformity of the femur in which the head is lower in relation to the great trochanter than normal, either from displacement downwards of the head on the neck, from curvature of the neck, or from a diminution of the angle between neck and shaft. In adults this angle is normally about 125°; in children it is 135°.

Etiology.—In young children coxa vara is in rare instances congenital; more commonly it is due to rickets. In most cases, however

COXA VARA

the infantile variety results from partial separation of the epiphysis of the head with an incomplete fracture of the neck due to injury, or from pseudo-coxalgia (q.v.). In *older children* the adolescent type is due primarily to old injury; and weight-carrying, repeated slight trauma, general debility, and a habit of standing in bad positions act as factors which lead to pronounced increase of the deformity, usually between the ages of 10 and 16. In *middle age* fracture of the neck, or in the region of the great trochanter, and osteo-arthritis of the hip cause coxa vara, while in the *aged* the neck bends as a result of senile bony atrophy.

Pathology. In the progressive stages the neck of the femur is rarefied as a result of chronic inflammation. The head is displaced downwards, and may become "mushroomed," and the neck is curved with a convexity forwards and slightly upwards. The angulation causes part of the head to lie outside the acetabulum. In late cases osteo-arthritis changes take place in the hip-joint.

Symptomatology.—Cases of the adolescent type are those which usually present themselves for treatment on account of symptoms produced by the coxa vara itself. There is pain, especially after exercise, the hip is held rather stiffly in walking, and there is a limp, the foot being rotated externally; in severe cases there is difficulty in kneeling and sitting. On inspection, slight wasting of the muscles of thigh and buttock is apparent, and the hip is in a position of adduction and external rotation. Secondary scoliosis is present, and produces great apparent shortening. On measurement the real shortening is rarely found to be more than $1\frac{1}{2}$ in.; estimation of Nélaton's line and Bryant's triangle shows that the trochanter is raised above the normal level. In bilateral cases there is lordosis, especially in the infantile variety of the disease.

All movements are diminished by muscular spasm in active cases, but this disappears after a few days' rest or under anaesthesia; it is then found that abduction is much diminished owing to the decreased angle between neck and shaft, internal rotation is lessened from the anterior convexity of the neck, and flexion is slightly less than normal; external rotation is much increased. In infants, however, particularly those suffering from rickets, movements are not restricted, because of the laxity of the capsule of the hip.

When osteo-arthritis is present the hip is in the typical position of coxa vara, but there is

diminution of all movements, thickening of the trochanter, and much wasting.

In some cases in children the anterior convexity of the neck is absent, and this variety is sometimes distinguished as *coxa adducta*.

Diagnosis.—In infants the condition must be diagnosed from *congenital dislocation of the hip*. Telescopic mobility and Trendelenburg's sign are absent in coxa vara, but the most important distinction depends on the presence of the head in its normal position in Scarpa's triangle, where it can be felt on rotation of the thigh; in congenital dislocation it can usually be felt in its abnormal position below and outside the anterior superior spine. In older children the deformity is liable to be mistaken for early *tuberculosis of the hip*. There is, however, less rigidity and wasting, and the hip is adducted and externally rotated, without flexion, instead of being flexed, abducted, and slightly externally rotated as in early tuberculosis; in the late stage of hip disease the limb is adducted with real shortening, but scarring, cold abscess, or sinus-formation is usually present, and there should be no difficulty in distinguishing the condition.

In all suspected cases, radiography will at once show the low relation of the head to the great trochanter. In cases of old standing it also shows, as a rule, secondary deposit of bone on the under aspect of the neck.

Prognosis.—In children the prognosis from conservative treatment is good, but it may be necessary to keep the weight off the affected limb or limbs for many months. In adolescence the less severe cases will recover gradually without operation, and in those with advanced deformity a good functional result may be expected from osteotomy. In elderly people with advanced osteo-arthritis little can be done for the coxa vara.

Treatment.—In the rickety and infantile forms of the disease the children must be prevented, by suitable splints, from walking or crawling; the hips should be massaged, and manipulated to a position of full abduction twice daily, the general treatment of rickets being also carried out.

Children over 3 years should be splinted in full abduction, and it may be necessary to begin by forcible wrenching, under anaesthesia, to obtain this position; a double Thomas hip-splint or a Jones abduction frame is then applied, and must be used, in combination with massage, for several months.

For a slight degree of coxa vara in older

children it is sufficient to take the weight off the affected limb by the use of a Thomas calliper splint, which transmits the weight from the great trochanter to the heel of the boot; the thigh is massaged and fully abducted daily, and the deformity gradually recovers in the course of several months. In severe cases the patient must be treated by traction of the thigh in a position of abduction, either by fixed extension on a Jones abduction frame or by weight-and-pulley extension, in which case the pelvis must be kept straight in bed by the application of a long Liston splint. After three months the deformity may be considered quiescent, the neck of the femur will be consolidated, and the patient may be allowed to walk in a calliper splint.

In adolescents and adults, when the disease has ceased to be progressive, osteotomy of the femur may be performed, either through or just below the trochanters, and will produce a good functional result in less time than does conservative treatment. After osteotomy the limb must be kept in full abduction until there is good union, and no weight must be borne on it for at least six months; during this time massage and movements should be continued.

C. W. GORDON BRYAN.

CRAMP.—A painful spasm, with temporary loss of function, of voluntary muscles. The causation is not always clear, but a want of balance between the amount of work performed by the affected group of muscles and their blood supply is the readiest explanation in most cases. This is obviously so in intermittent claudication or angina cruris, in which the supplying vessel is partially occluded. At rest the amount of blood going to the muscles is sufficient, but the extra supply which walking demands leads sooner or later to the characteristic painful tonus and limping. (*See also* ARTERIAL DEGENERATION.) Perhaps a spasm of the vessel-wall precedes the cramp in these cases, and is the sole cause in others, somewhat similar, for which no pathological basis can be found.

Exhaustion of the appropriate group of muscles is a recognized clinical antecedent, and its relation may be explained in the same manner. Cold appears to predispose. In the well-known and dangerous cramp of the thoracic muscles in swimmers there obtains a combination of cold, exhaustion, and the arduous use of muscles under conditions of difficulty, the intercostal muscles having the unwonted

duty of overcoming the pressure exerted by the water in addition to the usual atmospheric pressure, while the accessory respiratory muscles are actively engaged in swimming.

In some people cramp is induced with unusual ease, as by lying or sitting in constrained positions, sleeping upon an arm, etc. It frequently wakes the patient at night. The cramp which follows sitting on the hard, sharp edge of a chair indicates that in some cases compression of nerves (in this case the sciatic) is the exciting cause. A like result may follow tight bandaging, badly fitting splints, and the use of crutches.

A very painful form, affecting often the abdominal muscles and perhaps lasting for twenty-four hours or longer, has been described in those who work laboriously in a high temperature, e.g. stokers, workers in iron foundries, etc.

In cholera and other severe diarrhoeal disorders the peripheral cramps may, again, be attributed to defective circulation consequent upon the enhanced viscosity of the blood.

Among other causes of cramp are chronic gout, chronic interstitial nephritis, anæmia, Raynaud's disease, tetany, tetanus, strychnine poisoning, ergotism, and the group of painful paraplegias. Writer's cramp and other occupation cramps are considered under **NEUROSES, OCCUPATION**.

Treatment.—The treatment of particular forms is clearly that of their causes. The cramp occurring in people in good health, following exhaustion of the affected muscles, sleeping in bad positions, or arising spontaneously, is usually of very short duration. If time permits, massage, hot bathing, or rubbing with a rubefacient liniment will hasten recovery. Frequently recurring cramp at night may be prevented in many cases by the use of a hot-water bottle. A dose of antipyrin (10 gr.) on retiring is often efficacious in troublesome cases.

FREDERICK LANGMEAD.

CRAMP, WRITER'S, ETC. (*see* **NEUROSES, OCCUPATION**).

CRANIAL NERVES, LESIONS OF (*see* **NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF**).

CRANIOTABES.—A variety of defective ossification of the skull occurring in infants. Roughly circular or oval areas of unossified membrane exist in the parietal and occipital bones, and far less frequently in the

CRETINISM

frontal also. They are often remarkably symmetrical in their disposition, and are completely surrounded by ossified bone. They yield to the finger, and crackle like tightly stretched membrane. They are seldom noted after 6 months of age, and do not occur after the first year. Syphilis is an important cause; possibly they may also be due to rickets; and they are most marked when these two diseases are combined. The method of their production is undetermined, but they are thought to be due to osteoporosis from pressure and imperfect calcium metabolism.

A somewhat similar condition, erroneously called *craniotabes*, is more common. In it the bones are imperfectly ossified at their margins in the neighbourhood of the sutures and fontanelles. It occurs in rickets, in syphilis, in general malnutrition, and in states of high intracranial pressure, as in hydrocephalus and cerebral tumours, and may be found later than true *craniotabes*.

FREDERICK LANGMEAD.

CRETINISM.—A morbid condition of arrested mental and bodily development arising from an insufficient supply of thyroïdal secretion during the early years of life. This may be due either to congenital deficiency of the thyroid gland (congenital myxœdema) or to atrophic changes developing in it during infancy or childhood (juvenile myxœdema).

Etiology. Exciting causes.—Sporadic cases of cretinism occur in all parts of the world, but we do not know what is the exciting cause of the changes in the thyroid gland. Endemic cretinism occurs chiefly in narrow valleys in the mountains of Europe and Asia, and its distribution corresponds with that of endemic goitre. The evidence we possess shows that both these endemic maladies are due to some living agent which is conveyed by the drinking-water and is destroyed by boiling, the result of drinking the unboiled water being to produce goitre in the first generation and cretinism in the second or third.

Predisposing causes.—In congenital cretinism the signs are present at birth, but more commonly they appear during the first two or three years of life. Cretinism occurs nearly thrice as often in girls as in boys. It does not appear in two successive generations, as cretins are nearly always sterile. Two children may be afflicted in the same family, and the relationship to endemic goitre in the parent has been already mentioned.

Pathology.—Cretinism is the direct result of lack or destruction of the thyroid gland and the consequent diminution or failure of the supply of its hormones to the blood for distribution to the tissues. In congenital cases the thyroid gland has never developed, and no remains of the gland are found after death. In infantile cases the thyroid develops normally at first, but atrophies during infancy or early childhood, when the symptoms first appear. In these cases the gland is found to be diminished in size, the glandular tissue is degenerated, and the interstitial fibrous tissue largely increased in amount. In some cases cretinism has developed as the direct result of removal by operation or destruction by disease of a certain amount of the glandular tissue. In adolescent and adult cretins the thyroïdal insufficiency may be associated with goitrous enlargement and degeneration of the gland. Only some 8 per cent. of sporadic cretins have a goitre, whereas 66 per cent. of the 5,923 cases of endemic cretinism collected by the Sardinian Commission were found to be goitrous. The skeleton of the cretin is much less than the average for the same age. Adult cretins may be under 3 ft. in height and are rarely over 4½ ft. The long bones are short and thick, and may be deformed. The skull is short and broad, and the anterior fontanelle may remain open.

Symptomatology.—A typical cretin presents such a striking appearance that the nature of the malady is at once evident (Fig. 14). In the early stages the recognition of the disease is more difficult. At some period during the past few years of life it is noticed that the child is lacking in normal activity and that growth is slow. The face is pale and slightly swollen, the skin dry, the hair rather coarse in texture, and the extremities are cold. If treatment is started at this early stage the symptoms soon disappear, the child beginning to grow and becoming normal in appearance. If allowed to develop untreated, the condition gradually becomes intensified until all the typical signs of cretinism are present. A subcutaneous swelling extends all over the body; it is soft in consistence, but does not pit on pressure. It is especially abundant on the backs of the hands and feet and in the supraclavicular fossæ. The complexion is pale, and the skin dry and rough. The hair may be abundant in children but scanty in adults, the scalp being dry and covered with brown scales. The pubic and axillary hair is

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absent in the adult. The height is short, and may be less than 3 ft. at 28 years of age. The head is narrow in front and broad behind, and the anterior fontanelle long remains unclosed. Owing to the swelling of the subcutaneous tissues the face is full and rounded, with pendulous cheeks and chin, while the forehead may be

is marked lordosis of the lower dorsal and lumbar spine, with prominence of the abdomen, and an umbilical hernia is often present. The limbs are short, the hands and feet are broad and thick, and feel cold to the touch. The mental condition varies from slight mental hebetude in mild cases to idiocy in



Fig. 14.—A cretin. (Dr. Langmead's case. Photo: Dr. D. H. Paterson.)

wrinkled transversely. The eyes appear to be widely separated, the nose is short and broad, the tip being turned up so that the nostrils are directed forwards. The lips are thick and everted, the swollen tongue often being protruded between them. The teeth are poorly developed, and often carious. The neck is short and thick, and except in goitrous cretins no thyroid gland can be felt. There

severe ones. As a rule, cretins are slow in response and movement, placid in disposition, and sleep well. Owing to the lowered rate of metabolism the temperature is subnormal, and both pulse and respiration are slow. Anæmia and constipation are common. The generative organs and sexual characteristics remain undeveloped, so that in adult female cretins menstruation is scanty or absent.

CRETINISM

Diagnosis.—In an advanced case diagnosis is easy on inspection, but the early stages of the malady may readily be overlooked. Any arrest of growth or delay in development in a child should raise a suspicion of thyroidal insufficiency. In cases of doubt a course of treatment by thyroid extract should be given. If improvement follows, the diagnosis of cretinism is confirmed. *Mongolian idiocy* is apt to be mistaken for cretinism, but the mongol is more lively and observant, the cheeks are red, the palpebral fissure oblique, the epicanthic

thoroughness with which it is carried out. If the treatment is started early in life, as soon as the signs of cretinism are recognizable, and is adequately maintained and modified according to the growing requirements of the child, the outlook is good. Growth and physical development proceed in a normal manner, the signs of cretinism disappear, and the child becomes a normal adult. If the true condition of the child has been overlooked and development has been arrested for several years, the prognosis is less favourable. In



Fig. 15.—A mongol aged 10. (Dr. Langmead's case. Photo: Dr. D. H. Paterson.)

fold of skin is marked, and subcutaneous swelling is absent (Fig. 15). The tongue is pointed and is often sucked. Finally, only slight if any improvement takes place under the therapeutic test. An *achondroplastic dwarf* is characterized by marked shortness of the limbs relative to the length of the trunk. The skin, subcutaneous tissues, and hair are normal, and there is no arrest of mental or sexual development.

Prognosis.—Formerly the outlook for a cretin was miserable, for even if life was prolonged there was no prospect of improvement. Under rational treatment the prognosis now depends on the stage of the malady at which treatment is commenced and the

these cases physical development may be good and a normal standard may ultimately be reached. Mental development is usually slower, and is likely to fall short of the physical growth.

Treatment.—It must be clearly remembered and pointed out to the parents that the object of treatment is to supply the deficiency in the secretion of the thyroid gland in as nearly normal a manner as possible. The dosage of thyroid extract must therefore be proportionate to the amount of deficiency as shown by the severity of the symptoms and the varying need of the growing child. As the cretin has little or no thyroid gland of its own, the external artificial supply of secretion in the form of the

CUPPING

roid extract must be kept up for the whole of the patient's future life, and may be regarded more as a necessary part of the daily food than as a medicine. If the supply is discontinued the symptoms will inevitably return. Failure in treatment is usually due to a want of comprehension of the physiological principles of the treatment by those responsible for carrying it out. In a very young cretin, only very small doses of thyroid extract should be given at first. A sixth to a quarter of a grain of dry thyroid (thyroideum siccum, B.P.) or one or two minims of liquor thyroidei (B.P. 1898) may be given each night at bed-time. In older children double the dose may be given from the beginning. As the child grows the daily dose may be gradually increased to 2, 3, or 4 gr. of the dried gland in a powder or tablet, or to 5, 10 or 15 min. of the liquid preparation. Rapid loss of weight, restlessness, acceleration of the pulse, diarrhoea, and pains in the limbs are signs of overdosing. With suitable regulation of the dosage the subcutaneous swelling gradually disappears, the skin and hair become normal, and growth proceeds at the normal rate; in cases where growth has been arrested for some time it often proceeds at an abnormal rate, so that some of the loss is made good. Mental development proceeds, but may be backward, so that special education may be required in some cases. When the symptoms have disappeared and development is taking place at the normal rate, the dose should not be altered. As the child grows older, however, a slight increase in the dose may be required from time to time.

GEORGE R. MURRAY.

OROP (*see* DIPHTHERIA; LARYNGITIS; LARYNGISMUS STRIDULUS).

ORURA CEREBRI, LESIONS OF (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

ORURA CEREBRI, TUMOURS OF (*see* CEREBRAL TUMOUR).

CUPPING.—There are two forms of cupping, wet and dry, but the former is seldom used in the British Isles. The object of each is to bring blood to the surface, and in wet cupping this is allowed to escape through incisions made by a special scarifier. The incisions leave two rows of parallel scars, which are sometimes encountered during the examination of Russian sailors and others. Dry cupping is now employed chiefly in pleurisy and

CUT THROAT

in nephritis, especially when suppression of urine is present or threatened.

Method.—Special cupping glasses are sold, but a tooth glass or tumbler does equally well. As a preliminary precautionary measure it is advisable to mark out the area over which the cup is to be applied by a ring of vaseline; this will obviate burning and allow the cup to be removed more easily afterwards. Soak one or two small pieces of blotting-paper in methylated spirit and drop them into the glass, and then set them alight. When they are beginning to go out, "clap" the cup on to the vaselined ring. A somewhat better method is to put a few drops of the spirit into the glass and, by rotating it, to distribute them well over its interior. A pledget of cotton-wool may then be dipped in spirit and lighted and rubbed around the inside of the glass. This distributes the flame evenly over the inner surface of the glass. It is essential to make an unbroken circle of contact between the glass and the skin. **FREDERICK LANGMEAD.**

CUT THROAT.—The victim of a murderous assault upon the throat is usually beyond surgical aid; but the suicide, in his anxiety to accomplish his end, throws his head so far back that the great vessels gain protection behind the thick sterno-mastoids and so escape injury. (*See* INJURIES FROM THE MEDICO-LEGAL STANDPOINT.) After these self-inflicted wounds there are two dangers: hæmorrhage from veins or branches of the carotid artery that are severed, and asphyxia from blood trickling down the air-passages should these have been opened. The rules which govern **treatment** are simple. The patient is anaesthetized. If bleeding is profuse it must be stopped by digital compression while this is being done. After the surrounding skin has been cleaned, the clots are removed from the wound and the bleeding vessels ligated. The superior thyroid, lingual, or facial arteries, the external jugular, common facial, lingual, middle or superior thyroid veins are the vessels most likely to need tying. Severed muscles, including those at the base of the tongue, must be sutured. If the air-passage has been opened between the thyroid cartilage and the hyoid, this wound should not be sutured; it should be left freely open and the head kept in a slightly flexed position. If the larynx is opened through the thyroid cartilage or the crico-thyroid membrane a high tracheotomy should be performed. The thy-

roid cartilage or the crico-thyroid membrane should be sutured with catgut sutures and the overlying tissues similarly approximated. When the trachea has been opened the usually oblique wound must be carefully sutured with catgut, a vertical slit being cut downwards from the wound in front to accommodate a tracheotomy tube. Sepsis, being favoured by the accompanying anæmia, is likely to appear in these wounds. Secondary hæmorrhage is thus apt to occur.

C. A. PANNETT.

CURVATURE (*see* SPINAL CARIES; SPINAL CURVATURE).

OUTS, TREATMENT OF (*see* WOUNDS, TREATMENT OF).

CYANOSIS.—A lividity of the skin and mucous membranes. The blue tint is most evident at the periphery of the circulation—the fingers, toes, and ears—and parts which are highly vascular, and the superficial structures of which are thin, such as the face and lips.

Etiology.—There are two common causes of cyanosis—(1) defective circulation and consequent stagnation of the blood in the capillaries, (2) imperfect oxygenation of the blood (anoxæmia). Clearly these two are closely related, and frequently both factors are met with in the same case.

1. When the fault lies chiefly in **defective circulation**, it may depend upon (a) *cardiac causes*, as in failure of compensation: or atony, poisoning, or degeneration of cardiac muscle; (b) *constriction of arterioles*, as by exposure to cold, or in angio-neurosis and Raynaud's disease; (c) *concentration and increased viscosity of the blood*, as in severe diarrhœal conditions like cholera; (d) *obstruction to the venous return*, as by venous thrombosis or varicosity, or by pressure on veins produced by splints or tight bandages, masses of glands, neoplasms, etc.; (e) *paralysis of the vaso-motor centre*, as in meningitis, cerebral tumours, and other causes of increased intracranial pressure, and in affections of the bulb.

2. Cyanosis from **imperfect oxygenation** is very apparent in *congenital heart disease*, especially if pulmonary stenosis is present. It occurs with severe acute or chronic *pulmonary diseases*. Among the former, acute general tuberculosis of the pulmonary form is worthy of special mention, for in it the degree of cyanosis is very considerable and of diagnostic value; among the latter, fibrosis of the lung and emphysema are noteworthy, for in

them marked cyanosis may be compatible with a fairly active life. Into this group, too, fall *asphyxial states*, due to (a) *mechanical obstruction of the air-passages*, as in hanging, drowning, impaction of a foreign body; large masses of adenoids, retro-pharyngeal abscess, laryngeal diphtheria, œdema, ulceration, or growth, collections of mucus in the bronchi, as in whooping-cough, in bronchitis, and broncho-pneumonia, or pressure on the air-passages by a goitre, glands, tumours, etc.; (b) *imperfect action of the respiratory muscles*, as in compression of the chest, distension of the abdomen, paralysis of neural or central origin, or muscular spasm (tetanus, strychnine poisoning); (c) *inhalation of irritating fumes*, as those of ammonia or chlorine, or the gases used in war; or (d) *inspiration of an inadequate supply of oxygen*, as obtains at high altitudes, in the administration of nitrous oxide, or in confinement in an ill-ventilated or crowded room; (e) *paralysis of the respiratory centre* by drugs such as morphia, chloral, veronal, or by the toxins of disease, e.g. lobar pneumonia.

The cause of the peculiar heliotrope coloration in influenza during the epidemic of 1918-19 is unexplained.

Certain **rare forms** of cyanosis require brief descriptions:

Erythræmia (*syn. Vaquez's Disease, Polycythæmia vera, Splenomegalic Polycythæmia*).—There are three chief features of erythræmia—(1) increase of the red blood-cells, perhaps to 13,000,000 per c.mm.; (2) redness of the skin, especially that of the face, which changes to blueness in the cold or if the part observed is allowed to hang down; (3) enlargement of the spleen: this organ may reach to the pelvis, though generally its size is less than that seen in leukæmia; it is firm, uniformly enlarged, and free from tenderness. Other symptoms which may be present are headache, giddiness, dyspnoea, and pains in the limbs. The etiology is obscure. Hyperplasia of the bone-marrow has been found after death.

Sulph-hæmoglobinæmia.—The cyanosis in this condition is of a greyish colour and is accompanied by pallor. The patients complain of great weakness, and there may be prolonged periods of severe prostration. A history is usually obtainable of constipation of many years' standing, perhaps alternating with bouts of offensive diarrhœa. The disease is due to the combination of sulphur with the hæmoglobin in the red corpuscles, and it may possibly be brought about by defects in the

intestinal mucous membrane, or by bacteria. Diagnosis is only possible with the help of the spectroscope. If the intestinal condition reacts to treatment the prognosis is not hopeless.

Methæmoglobinæmia.—The cyanosis is of a brighter blue than in the former condition, but is also associated with pallor, offensive diarrhœa, and weakness. These are attacks of alarming dyspnœa; on the other hand, there may be periods of freedom from cyanosis. Dimness of vision is sometimes complained of. Methæmoglobinuria occurs.

Etiologically, cases of methæmoglobinæmia may be separated into two categories: (1) those due to drugs or poisonous fumes, and (2) enterogenous cases. Among the drugs which produce the condition are the aniline derivatives (antipyrin, acetanilide, phenacetin, exalgin), the benzene compounds, and potassium chlorate. There is usually a history of addiction to one of these drugs, of overdosage, or of employment in their manufacture. The enterogenous cases are possibly of bacterial origin. The final diagnosis rests on the spectroscopic examination of the blood.

Treatment.—This is essentially that of the cause, and will be found under the conditions mentioned. In heart disease, and in acute pneumonia, cyanosis, if marked, is an indication for venesection. Oxygen inhalations often give considerable temporary relief to the discomfort arising from the cyanosis itself. For the enterogenous cases, constipation must be overcome, and treatment with intestinal antiseptics may be attempted.

FREDERICK LANGMEAD.

CYCLICAL VOMITING (see VOMITING, CYCLICAL).

CYELITIS (see UVEAL TRACT, AFFECTIONS OF).

CYSTIC HYGROMA.—One of the varieties of cavernous lymphangioma, a cystic hygroma is a subcutaneous or intermuscular new formation of lymphatics, dilated into cystic processes. The lobules have thin walls lined by a single layer of lymphatic endothelium, contain clear fluid, and are loosely connected by fibrous and fatty tissue, which is continuous with adjacent fasciæ and vessel sheaths. The tumour is very liable to attacks of inflammation, after each of which it shrinks, and it may eventually disappear spontaneously, leaving a small amount of scar tissue.

Symptomatology.—The tumour is often

present at birth and increases in size during early infancy; common situations are the posterior triangle of the neck, the axilla, and the back. A soft multilobular swelling is palpable, of very indefinite outline, giving the sensation of a cyst only partially filled with fluid. The skin may be stretched over the tumour, has a bluish appearance, and often presents dilated blood-vessels. Processes of the swelling extend deeply under neighbouring muscles. Attacks of inflammation are accompanied by fever, local redness, and increase of the swelling. Spontaneous suppuration is very rare.

Diagnosis.—A *lipoma* is uncommon in a young child, and has a firmer consistency and more definite outline. The absence of surrounding infiltration will serve to distinguish an inflamed cystic hygroma from an *acute abscess*.

Treatment.—Spontaneous cure is so common an occurrence that in infancy no treatment is advisable unless the tumour is growing rapidly. Later on, if it persists or increases, excision may be practised, but it is not to be undertaken lightly; removal involves a difficult dissection with free bleeding, and there is a special risk of dangerous suppuration.

C. W. GORDON BRYAN.

CYSTIO TERATOMA (see OVARIAN CYSTS).

CYSTINURIA (see URINE, EXAMINATION OF).

CYSTITIS.—Inflammation of the bladder, which may be either acute or chronic.

Etiology.—With the exception of those few instances in which the application of too concentrated solutions of chemical irritants has proved the exciting cause, cystitis is always primarily bacterial in origin. The chance infection with pathogenic micro-organisms of the urine contained in a healthy bladder, as after the passage of a septic catheter, is not alone usually sufficient to produce cystitis, the bacteria being washed out naturally during the next act of micturition before any damage or absorption can take place. This fact has been demonstrated repeatedly by experiments on animals; in them, in addition to introducing bacteria, it is necessary to injure the bladder-wall in some way, as by bruising or by inducing artificial retention of urine, before inflammation and invasion of the mucous membrane can occur. When, however, large numbers of pathogenic organisms repeatedly enter the bladder with each ureteric efflux, as in primary

CYSTITIS

infection of the kidney with *B. coli* or the tubercle bacillus, they alone appear to be sufficient to determine the onset of cystitis after a time.

Two factors, then, are usually essential for the production of cystitis—(1) some condition producing impairment of the natural vital activity of the bladder-wall; (2) the presence of an infecting agent.

1. Predisposing causes.—(i) Any disease, such as enlargement of the prostate, stricture, or atony of the bladder, which prevents the latter from emptying itself completely at each act of micturition. Certain organic nervous diseases, such as a transverse myelitis or tabes dorsalis, also come into this category, but here there is actually a trophic disturbance of the bladder-wall in addition. In women the residual urine occurring in cases of cystocele is a splendid medium for the growth of organisms, and pregnancy also appears to have some important causal relationship.

(ii) Traumatism, e.g. injury to the bladder-wall by the careless passage of a catheter or sound, or bruising occurring during labour or during the course of abdomino-pelvic operations.

(iii) The presence of a stone or foreign body.

(iv) New growths of the bladder. It is unusual to find cystitis in cases of simple pedunculated papillomata, but where the change is a diffuse villous growth affecting a considerable area of the bladder-wall, or in malignant disease, a sloughing condition of the growth with foul septic urine is an invariable accompaniment of the later stages.

2. The infecting agent.—The commoner pathogenic organisms found in cases of cystitis may be classified as follows:—

(i) Those present in acid urine: *B. coli*, the tubercle bacillus, and the gonococcus.

(ii) Those present in neutral or faintly acid urine: *B. coli* and streptococci.

(iii) Those present in alkaline urine: staphylococci and proteus. These two organisms possess the power of splitting up urea into simpler ammonium salts and rendering the urine alkaline.

Methods of infection. (1) *From above.*—Direct invasion of the bladder-wall by organisms carried down to it in the stream of urine from primary renal infections of *B. coli* or *B. tuberculosis*.

(2) *From below.*—(a) The passage of a septic catheter or instrument. (b) The upward spread by direct continuity or by the lymph-stream

of the mixed organisms of a posterior urethritis, or of tuberculosis or other infections of the prostate gland.

(3) *Primary infection of the bladder.*—(a) Invasion of the bladder-wall by *B. coli* or other organisms by direct spread through the walls of the large intestine, or from a pyosalpinx or pelvic abscess. (b) By the blood-stream, e.g. in tuberculosis (very rare).

Pathology.—The mucous membrane of the bladder shows the changes usually seen in catarrhal inflammation of a mucous membrane, and secretes large amounts of thick, ropy mucus. In very acute cases there may be extensive sloughing and exfoliation of the mucous membrane, and in the more prolonged and severe cases of tuberculosis or other chronic infections the smooth lining of the bladder may be almost entirely replaced by a layer of easily bleeding granulation tissue. The muscular wall undergoes great hypertrophy and contracts, so that in extreme cases the bladder is physically incapable of holding more than $1\frac{1}{2}$ or 2 oz. of fluid, apart from actual irritability.

The cystoscopic appearances are described in the next article.

Symptomatology. (1) *Acute cystitis.*—An attack of acute cystitis begins with a feeling of suprapubic discomfort, which, within an hour or two, becomes a severe ache accompanied by an intense desire to micturate. Micturition may be as frequent as once every ten minutes, a few drachms only of cloudy urine, which in the more virulent coliform infections may be bloodstained, being passed with much scalding pain in the urethra on each occasion. Sometimes a few drops of bright blood escape with the last of the urine. As micturition is completed, the patient may suffer most acutely from violent cramp-like pains in the region of the bladder-neck. Sleep is impossible, and the patient lies exhausted, awaiting with the greatest dread the next call to micturate. The mucous membrane of the bladder does not appear to be capable of much absorption, and, in the absence of renal infection, the temperature rarely rises above 100° or 100.5° F.

After a few days of intense and distressing pain the symptoms of an acute attack generally subside, and may disappear completely within the course of a few weeks. Occasionally these cases drag on for many weeks or months with little or no improvement and gradually pass into a chronic condition.

CYSTITIS

(2) **Chronic cystitis.**—This may be the sequel to an acute attack or may develop insidiously. Beyond some increased frequency of micturition and vague feelings of suprapubic discomfort, the patients affected in this way appear to suffer little inconvenience from their complaint. The capacity of the bladder, however, steadily diminishes, and, even after all evidence of infection has disappeared, the increased frequency of micturition due to the contracted bladder will remain, and may prove most difficult to treat. The urine varies in appearance from haziness only, as seen in the terminal bacillurias of the coliform infections, to the thick, foul urine of prostatic and stricture cases, but remains fairly constant from day to day. If it is allowed to stand, a thick tenacious mass of muco-pus quickly settles to the bottom of the specimen glass, in marked contrast to the more diffused and flocculent deposit seen in pyurias of renal origin.

Treatment. (1) **Acute cystitis.**—The patient should be put to bed at once, and restricted to a milk diet. He should be encouraged to drink large quantities of barley water and of the natural aperient waters to flush out the kidneys and bladder and render the urine as non-irritating as possible. Hot fomentations to the lower abdomen or, better still, repeated and prolonged hot hip-baths afford the greatest relief, and suppositories of morphia and belladonna will usually be found most useful in helping to control the pain. If the patient is suffering from want of sleep, a hypodermic injection of heroin, $\frac{1}{4}$ gr., should be given without hesitation.

Occasionally autogenous vaccination appears greatly to relieve the pain; this usually coincides with marked improvement in the temperature chart. In all probability this form of treatment, although it appears to alleviate, does not actually shorten the natural course of the disease.

The following mixture, administered every four hours, will be found useful:—

℞ Acid. bor. gr. x.
Tr. hyosey., gr. xx.
Inf. buchu ad ℥i.

Until the acuteness of the attack has completely subsided, instrumentation of any kind whatsoever must most studiously be avoided.

(2) **Chronic cystitis.**—When all acute symptoms have disappeared, every effort should

be made with the help of the cystoscope to exclude any predisposing cause to infection. A vesical stone should be crushed and the obstruction due to an adenoma of the prostate or urethral stricture dealt with. In very old-standing cases it may be found advisable to combine the appropriate treatment of these conditions with a preliminary suprapubic drainage and daily lavage of the bladder for a time. It is of vital importance also to identify the presence of any renal infection, if it be present, as it is of little use to confine the attention to cleansing the bladder if infected urine from a chronic pyelitis is continually entering that viscus by the ureteric openings. The early symptoms of primary renal infections (*B. coli* or *B. tuberculosis*) may be almost entirely vesical, and the site of the original lesion will be missed if a careful examination of the ureteric orifices is neglected. At the same time it should be noted that a coliform infection of the bladder may remain, and resist all forms of treatment, long after the original pyelonephritis has completely resolved.

In cases of chronic cystitis, excluding those due to tuberculous disease, the bladder should be thoroughly washed out once or twice daily with one of the following solutions:—

(1) Boric acid, gr. x-xx ad ℥i.

(2) Oxycyanate of mercury, 1 in 8,000 to 1 in 4,000.

(3) Silver nitrate in distilled water, gr. $\frac{1}{4}$ — $\frac{1}{2}$ ad ℥i.

Boric-acid lotion will be found most suitable for the subacute cases, whilst in the chronic cases the best results will be obtained by employing oxycyanate of mercury and silver nitrate solutions for alternating periods of five or six days each.

The following mixtures for internal administration may assist:—

(1) For cases with foul ammoniacal urine:

℞ Urotropine gr. x.
Sod. benz. gr. xx.
Inf. buchu ad ℥i.

One ounce every six hours.

This should be combined with acid sodium phosphate, gr. xv, given night and morning.

(2) For acid pyurias:

℞ Salol gr. v.
Acid. bor. gr. x.
Inf. buchu ad ℥i.

One ounce every six hours.

If a case of chronic cystitis does not improve

CYSTOSCOPY

when treated on the above lines, the bladder should be drained for two to three weeks by means of a large rubber tube inserted through a suprapubic cystotomy wound, and lavage carried out through this tube instead of by the urethra. Autogenous vaccination is useless in the treatment of these cases.

HAROLD W. WILSON.

CYSTOOCELE (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

CYSTOSCOPY.—Inspection of the interior of the bladder through the urethra is termed cystoscopy. There are two methods of cystoscopy, the *open* or *direct* and the *indirect*.

Direct or **open cystoscopy** was introduced by H. A. Kelly, and consists in distension of the bladder with air by atmospheric pressure and examination through a straight open tube by means of reflected light or an electric hand-lamp. Kelly's speculum (Fig. 16) is a plated metal cylinder $3\frac{1}{2}$ in. long with a funnel-shaped expansion at the outer end, a handle attached at a right angle, and an obturator.

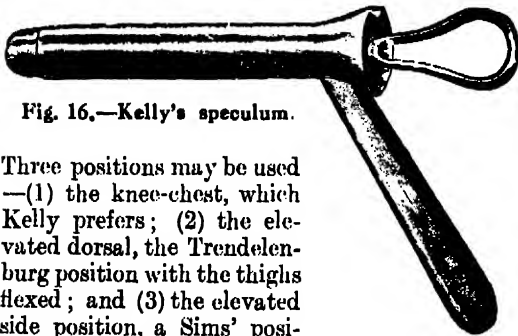


Fig. 16.—Kelly's speculum.

Three positions may be used—(1) the knee-chest, which Kelly prefers; (2) the elevated dorsal, the Trendelenburg position with the thighs flexed; and (3) the elevated side position, a Sims' position with the table raised.

Local or general anaesthesia may be used, and it may be necessary to dilate the urethral meatus with a calibrator. The speculum is introduced and the obturator removed, and any urine remaining in the bladder is allowed to escape. The light is now projected into the bladder and the interior is inspected systematically. A long, fine rubber tube with a suction bulb at the outer end is used to remove fluid which accumulates during the examination.

Indirect cystoscopy is performed by means of a cystoscope, first introduced by Nitze. In its simplest form the cystoscope consists of an optical apparatus and an illuminating apparatus combined. The optical apparatus is com-

posed essentially of three lenses, which give an erect and slightly enlarged figure. The window at the vesical end of the cystoscope looks out at right angles to the axis of the tube in which the lenses are set, and in order to deflect the image along the tube a prism is placed at this end of the ocular system. The illuminating apparatus consists of a small metal filament incandescent lamp in a metal hood, set at an angle to the shaft of the instrument, which illuminates the field to which the window of the cystoscope is directed.

Connexion with the source of the electric current is obtained by flexible insulated wires and a metal contact.

In order to avoid the preliminary introduction of a catheter and to facilitate washing the bladder during examination, the cystoscope shown in Fig. 17 has been introduced. In this the illuminating apparatus is in the form of a tube bearing the lamp at its extremity, and the optical apparatus is a telescope which can be inserted into this when the necessary irrigation and distension of the bladder have been completed. The most convenient source of electricity is a small dry-cell battery of $4\frac{1}{2}$ or 8 volts. When the main electric current is used, a fixed rheostat or a transformer is necessary, and a pantostat, when available, is to be preferred.

A special cystoscope is required for catheterization of the ureters (Fig. 18). It consists of an irrigating cystoscope with a tunnel on the upper surface along which the ureteric catheter passes. This tunnel stops short of the window on the concave surface of the beak, and an elevator worked by a screw at the ocular end is used to raise the point of the catheter and facilitate its introduction into the ureteric orifice.

Method of performing cystoscopy.—The patient is examined in the lithotomy position, the surgeon standing to pass the instrument and wash the bladder, and sitting opposite the perineum for the examination. In the majority of cases a local anaesthetic only need be used. In exceptional cases, such as very nervous individuals, young girls, children, and where the bladder is unusually sensitive, as in tuberculous cystitis, a general anaesthetic may be used. Spinal anaesthesia is also useful in difficult cases. Local anaesthesia is produced by instilling 30 or 60 minims of 5-per-cent. novocain solution into the male anterior urethra by means of a glass pipette with a small rubber bulb, and massaging i

CYSTOSCOPY

back along the urethra into the prostatic urethra. A Guyon syringe and catheter may be used to introduce the novocain solution accurately into the prostatic urethra. The female urethra seldom requires an anæsthetic.

A small enema of hot water containing 20 gr. of antipyrin, or, in the female subject, a hot vaginal douche containing 30 gr. of antipyrin, given half an hour before the examination, will relieve spasm in a hypersensitive bladder.

The best lubricant is glycerin, but liquid paraffin may be used as a substitute.

der syringe made for me by Messrs. Weiss. About 8 or 10 oz. of sterile water, saline solution, or boric-acid solution should be introduced.

The bladder should be examined systematically. The window is turned downwards so that the base of the trigone or interureteric bar, a transverse ridge, comes into view. If this is followed to each side, a small red slit, the ureteric orifice, will be seen, and diastole and systole of the muscle surrounding the orifice may be observed at intervals of some seconds, and the swirl of an efflux of urine noted. In the trigone the mucous membrane is darker

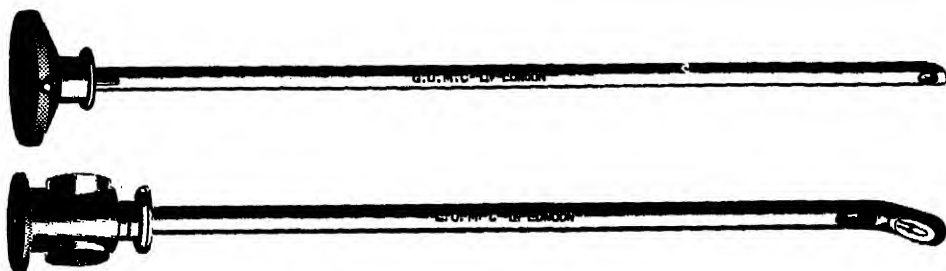


Fig. 17.—Examination cystoscope. (Author's model)

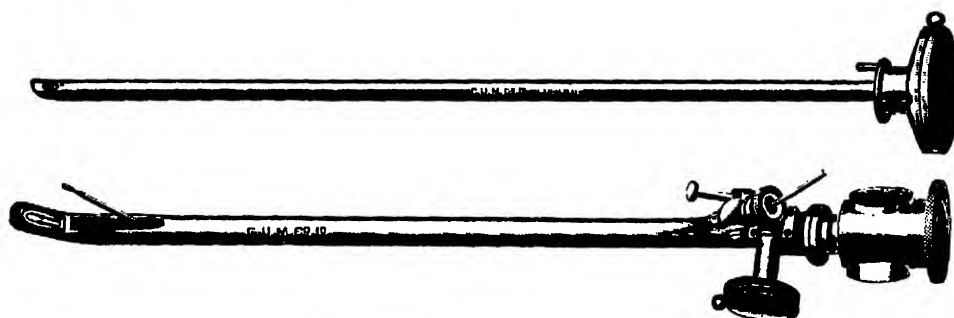


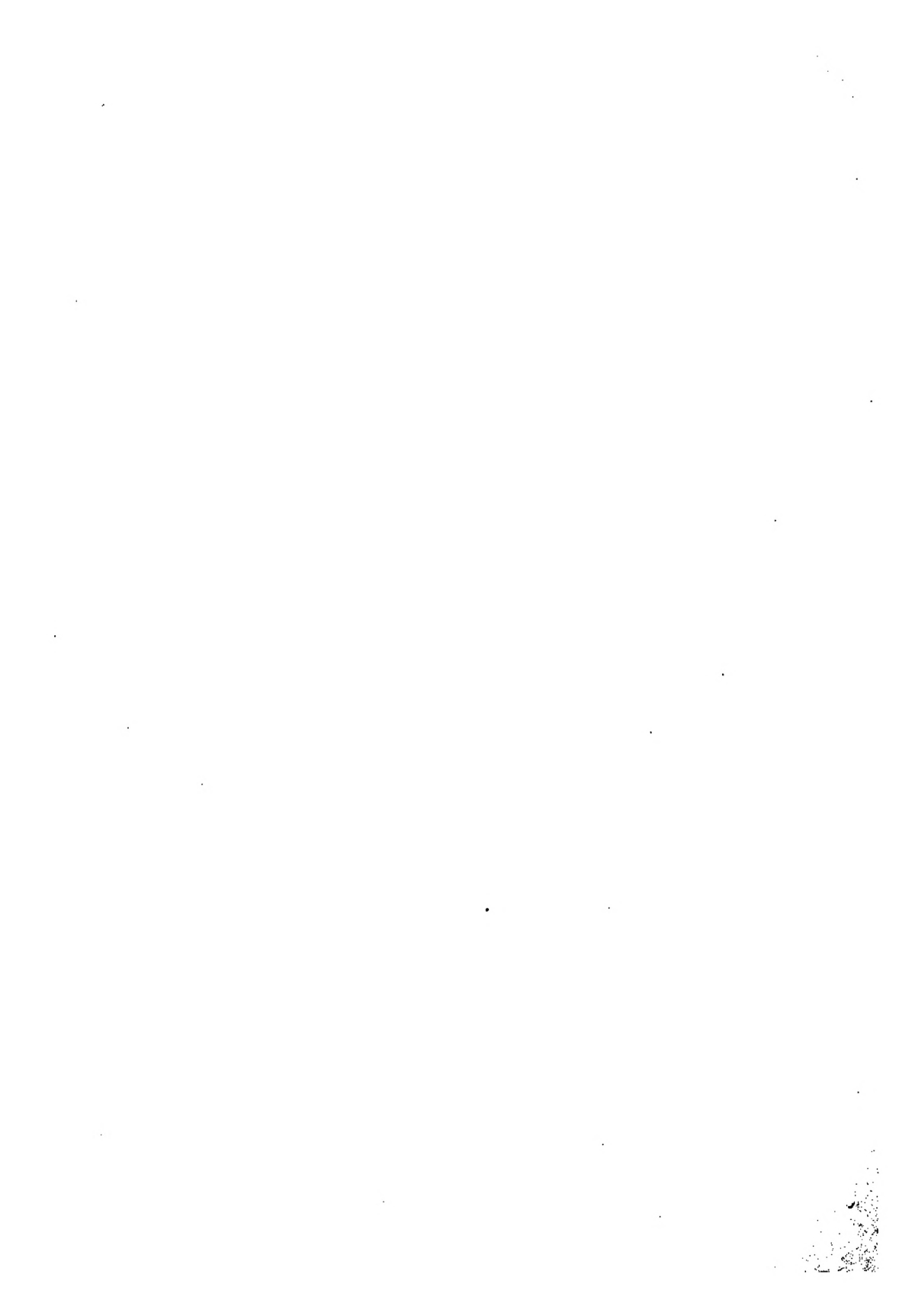
Fig. 18.—Catheterization cystoscope. (Author's model)

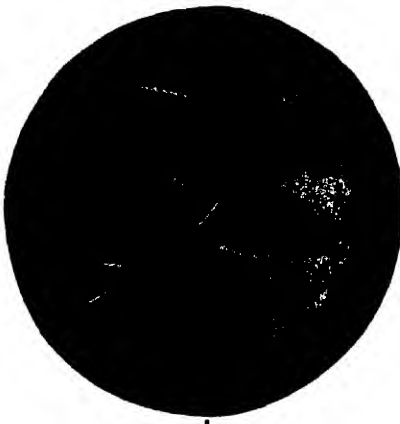
After the urethra has been anæsthetized, the irrigating cystoscope is passed. In the male the beak sinks down 5 or 6 in., and then the outer end of the cystoscope is depressed so that the beak engages in the membranous urethra. Further depression and pushing the instrument on will make it ride over the prostate. The telescope is now withdrawn, the drainage spigot inserted, the urine drains away from the bladder, and its translucency is tested by examining it in a clear glass. If it is murky with blood or pus, washing of the bladder is required. For washing and filling the bladder, a bladder syringe or an irrigator may be used. I use an 8-oz. all-metal blad-

der syringe made for me by Messrs. Weiss. About 8 or 10 oz. of sterile water, saline solution, or boric-acid solution should be introduced.

The posterior, the lateral, and the anterior walls are now inspected, and the fine, sandy, light-reflecting appearance with delicate vessels is noted. At the apex an air-bubble introduced with the cystoscope is seen. Lastly, the edge of the urethral orifice is examined by withdrawing the cystoscope till the window begins to sink into the urethra. The edge is smooth and slightly concave.

Abnormal openings in the bladder-wall may be fistulæ or diverticula. A *fistula* may result from the rupture of an abscess into the bladder, or be a communication between the bladder





1



2



3



4



5



6

1, Trabeculated and sacculated bladder. 2, Haemorrhagic cystitis. 3, Acute cystitis.
4, Tuberculous cystitis. 5, Malignant growth. 6, Papilloma.

PLATE 8.—CYSTOSCOPY.

CYSTOSCOPY

and the bowel, or may open on the skin surface or into the vagina. In an entero-vesical fistula there may be a small area of cystitis surrounding the opening, and the rest of the bladder may be healthy, but frequently the bladder shows marked cystitis, and the orifice of the fistula is hidden by folds of mucous membrane.

A *diverticulum* shows a solitary opening, or sometimes multiple openings, round and dark, with some trabeculation surrounding the orifice.

Cystitis may be localized or general, acute or chronic. In the majority of cases the cystitis affects part of the bladder, usually the base, and general cystitis is less common. In acute cystitis the mucous membrane is intensely red and has a non-reflecting spongy appearance, the blood-vessels have disappeared, and flakes of mucus adhere to the surface (PLATE 8, Fig. 3). Cystitis which is chronic from the clinical point of view may retain the cystoscopic appearances of acute cystitis. In very old-standing cystitis the mucous membrane is dull-reddish, brown, or greyish, and the blood-vessels have disappeared. Trabeculation and sacculation may be found if obstruction is present (PLATE 8, Fig. 1). The following names are applied to special types of cystitis, viz. papillary or vegetating, phosphatic, bullous, cystic, granular, leukoplakic. Hæmorrhagic cystitis is a very acute form seen in mixed infections (PLATE 8, Fig. 2).

Ulceration of the bladder (non-tuberculous) may be superficial or deep. Superficial ulcers are irregular and elongated, and lie on the crests of trabeculae and folds of mucous membrane. Deep ulcers may be multiple or single, irregular or round. In tuberculosis of the bladder the organ is highly sensitive and its capacity diminished, and slight persistent oozing of blood sufficient to obscure the cystoscopic view is common. Tubercles (PLATE 8, Fig. 4) are found fairly frequently lying round a ureteric orifice, in the neighbourhood of an ulcer, or scattered over the mucous membrane in groups. Tuberculous ulceration is superficial or deep. Radiating lines of puckered mucous membrane round the irregular superficial ulcers are characteristic. Deep tuberculous ulcers have an irregular undermined edge.

Calculi and *foreign bodies* form easily recognized objects in a cystoscopic examination.

In the most frequent form of *tumour* of the bladder—the villous growth or papilloma—the appearances vary greatly. The commonest form resembles a tiny pink sponge. Close in-

spection shows densely packed finger processes or villi, in each of which is seen a looped blood-vessel. In a less frequent variety the villi form loose, wavy tendrils (PLATE 8, Fig. 6). Papillomata are single or multiple, small and large. Malignant growths may resemble simple papillomata, but are sessile, the villi being stunted and so closely packed that the surface has a smooth, raspberry-like appearance (PLATE 8, Fig. 5). The mucous membrane at the base may be rigid and puckered, and may bear oedematous tags. Nodular growths vary in size from a pea to a Tangerine orange. The surface is smooth, nodular, opaque, and pink or yellowish in colour. Infiltrating growths form flat nodules on the mucous membrane, and there may be a central depressed area, with a raised hard ring of nodular growth.

Ureteric orifices and catheterization of the ureters.—If there is any difficulty in finding the ureteric orifice, an intramuscular injection of indigo-carmin may be given, when the dark-blue efflux can be easily seen and the orifice discovered.

A double ureteric orifice may be present where a double ureter and renal pelvis exists. An area of inflammation surrounds the ureteric orifice in pyelitis. Prolapse of the ureter due to a narrow orifice produces a cyst-like body which becomes distended when the ureter contracts. A stone may be found projecting from the ureteric orifice.

Catheterization of the ureters is performed for various objects. The preparation is similar to that for ordinary cystoscopy. After the bladder has been filled and the telescope introduced, a fine ureteric catheter is pushed along the catheter tunnel until the end lies just within the field of the window. The ureteric orifice is found and the window of the cystoscope manoeuvred close to it. The point of the catheter is now elevated by turning the screw at the ocular end, and slipped into the orifice. The catheter is pushed gently onwards. In my pattern of ureteric catheter each half-inch is brown and black alternately, and the 6-in. stage is marked by a narrow red band, the 12-in. by two red bands. When the 12-in. mark is at the ureteric orifice, the eye of the catheter is in the renal pelvis. If it is intended to catheterize both ureters, a double barrelled cystoscope is used, or a single-barrelled cystoscope may be withdrawn, leaving the first catheter in position, and reintroduce alongside it loaded with another catheter.

The urine is allowed to drip into stand

DACRYOCYSTITIS

bottles or test-tubes. The amount collected depends upon the nature of the examination required. Two or three drachms will suffice for an ordinary bacteriological examination; an ounce or preferably several ounces should be collected for examination for tubercle or for the estimation of urea.

The following are the chief uses of ureteral catheterization: (a) *Diagnosis*: (1) The presence and functioning of a kidney. (2) Is the kidney or bladder infected? (3) Which kidney is affected? (4) Is the kidney dilated? (5) Sounding the ureter. (6) Indicating the course of the ureter. (b) *Treatment*: (1) Lavage of the renal pelvis. (2) Injection of oil, etc., in descending ureteral calculus. (3) Dilatation of stricture of the ureter.

The functioning of the kidney is examined by (a) estimating the urea in a specimen of urine obtained by ureteral catheter, (b) testing the excretion of coloured substances by the kidney. The best colour test is the phenol-sulphone-phthalein test introduced by Geraghty. The patient drinks 400 c.c. of water, and twenty minutes later 1 c.c. of fluid containing 6 mg. of phenol-sulphone-phthalein is injected into the muscle of the buttock. The bladder is emptied and the catheter retained, the urine

being allowed to drip into a bottle containing a few drops of sodium hydrate. A bright purple-red colour appears when the excretion begins. The urine is collected for an hour, and separately for a second hour. Each urine is diluted to one litre and the percentage of colouring matter estimated by a colorimeter. In normal cases the colouring matter appears in from five to ten minutes. Forty to 60 per cent. of the drug is excreted in the first hour, 20 to 25 per cent. in the second hour, and 60 to 80 per cent. in the two hours.

J. W. THOMSON WALKER.

CYST-CONTENTS, EXAMINATION OF (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

CYSTS (see under individual organs).

CYSTS, BRANCHIAL (see BRANCHIAL CYSTS).

CYSTS, DERMOID (see DERMOID CYSTS).

CYSTS, HYDATID (see HYDATID DISEASE).

CYSTS, SEBACEOUS (see SEBACEOUS CYSTS).

CYTO - DIAGNOSIS (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

DACRYOCYSTITIS.—Inflammation of the lachrymal sac is usually primary, but may be secondary to obstruction in the nasal duct. When primary, the inflammatory swelling of the sac-wall causes blockage of the upper end of the nasal duct.

Etiology.—Primary inflammation of the sac may be due to microbic infection spreading from the conjunctiva or from the nasal mucous membrane. In some cases, especially in children, the condition is *tuberculous*, with much thickening of the sac-wall, and occasionally caries of the subjacent lachrymal bone. In trachomatous districts, *trachomatous* infiltration of the sac may be met with.

The causes of obstruction in the nasal duct are:—

1. *Within the lumen*:

- (a) Congenital membrane.
- (b) Epithelial debris in the newborn.

(c) Foreign bodies, such as a leaden style which has slipped down from the sac out of reach.

2. *In the wall of the duct*:

- (a) Cicatricial tissue from preceding inflammation, from false passages made in passing a probe.
- (b) Inflammatory hypertrophy of the nasal mucous membrane in the inferior meatus.

3. *Outside the duct*:

- (a) Periostitis of the neighbouring bones, commonly syphilitic, congenital or acquired.
- (b) Injuries of the bony wall of the duct, especially gunshot wounds.
- (c) Malignant or gummatous ulceration of the nasal inferior meatus.
- (d) Distension of the inner wall of the maxillary antrum by cyst or malignant growth.

DACRYOCYSTITIS

Symptomatology.—The disease is essentially a chronic one, on which acute symptoms are sometimes superimposed. The patient complains of epiphora (i.e. overflow of tears), usually of long standing, especially when out of doors. There is some crusting of the lid margins on awaking after sleep, and there may be an obvious swelling in the neighbourhood of the inner canthus (mucocele of the sac). The sac contains mucoid or purulent fluid, which can be made to regurgitate through the upper or lower punctum by pressure with the finger just below the internal tarsal ligament behind the bony orbital margin.

In some cases the nasal duct is only partially blocked, and pressure on a mucocele causes the contents to pass downwards to the nose. In other cases there is obstruction of the nasal duct, but no distension of the sac; such cases commonly develop a mucocele later. In other cases, again, fluid may be pent up in a diverticulum of the sac, and will not regurgitate on pressure.

Diagnosis.—Apart from superficial skin conditions, such as sebaceous cyst, mucocele of the sac has to be diagnosed from mucocele of the anterior ethmoidal cells, dermoid cyst, and meningocele. *Meningocele* is met with in the upper and inner angle of the orbit, not below the internal tarsal ligament. *Dermoid cysts* are more common in the outer parts of the eyelids, whilst a *mucocele of the ethmoidal cells* comes forward most commonly above the internal tarsal ligament. Mucocele of the sac reaches but rarely above this ligament, and in cases of doubt the diagnosis will be confirmed by syringing: if the duct is clear, ethmoidal mucocele is suggested; if it is blocked, there is dacryocystitis. It is wise to make a rhinological examination in all cases.

Treatment.—In a recent case the lumen may sometimes be restored by keeping the interior of the sac clear of pus. This is attempted by instructing the patient to empty the sac by finger pressure on the inner canthus, previous to irrigation of the conjunctiva with boric-acid lotion; the sac is syringed daily by the surgeon.

The instruments required for *syringing the lachrymal sac* are a conical punctum dilator and a "record" syringe with blunt lachrymal nozzle. A 2-per-cent. solution of cocaine is applied to the conjunctiva, or, if complete anaesthesia is desired, a few crystals of cocaine hydrochloride may be rubbed on the lid margin

internal to the punctum. The surgeon stands behind the patient, and stretches the lower lid by a finger applied to its outer part, so as slightly to evert the inferior punctum, into which the point of the dilator, held vertically, is introduced. As soon as it is engaged, the dilator is brought to the horizontal, its point remaining in the punctum, and is then pressed gently inwards towards the nose along the canaliculus by rotary movements until the punctum is deemed to be sufficiently dilated to admit the nozzle of the syringe. The syringe is filled with warm lotion and its nozzle introduced along the canaliculus; when it is well inserted the piston is pressed and the sac irrigated. If the fluid meets with resistance, the point may have caught in a fold, and the nozzle should be withdrawn slightly.

When the sac has been cleared, a few drops of 10-per-cent. argyrol may be introduced by the same method; but if there is any chance of the mucous membrane having been torn, this should be omitted, or a permanent "black eye" may result.

Such treatment may be persisted in for a week or two. If unavailing, further measures may be taken.

The *passage of probes* is especially indicated in congenital cases, in which one operation may produce a cure. The sac is first cleansed by syringing, and some drops of 4-per-cent. cocaine are left in the sac for a few minutes; a small Bowman's probe is then introduced horizontally through the lower canaliculus; by rotary movements its point is passed on into the sac, when it should be felt to tap against the bone covered only by the thin sac-wall. Using the point as a centre, one brings the probe to the vertical, and the point is passed gently downwards into the duct. In this small operation, care must be taken not to make a false passage. If moderate pressure does not suffice, one must either desist, or slit up the canaliculus with a probe-pointed Weber's or Tweedy's knife and pass a Stilling's knife into the duct in order to divide the constriction. Larger-sized probes may then be passed.

The operation is a painful one, and, except in congenital cases, usually requires repetition. Even then the proportion of successful results is by no means high. For this reason not a few surgeons have given up the procedure.

A case in which the stricture tends to close

DACRYOCYSTITIS

after probing may be treated by leaving a lead or silver style in the duct for some weeks or months.

If a more rapid cure is necessary in old-standing cases, the patient will be well advised to submit to the operation either of *excision of the sac* itself or of intranasal drainage—e.g. *West's operation*. The results of the former are usually very satisfactory, the patient only suffering from epiphora when exposed unduly; the results of the latter operation have in some hands given good results.

Complications.—1. **Acute dacryocystitis** may arise at any time by the passage of micro-organisms into the surrounding tissues. There is a tender swelling in the sac region with œdema of the lids, most pronounced below the inner canthus. If inflammation persists the skin becomes involved, and an abscess forms which eventually discharges itself just below and externally to the inner canthus. Through such an aperture the sac may continue to empty, with the formation of a lachrymal fistula. If the aperture heals, there may be frequent relapses of inflammation. The condition has to be diagnosed from *facial erysipelas*. The œdema of the lower eyelid in the latter condition often has a raised edge opposite the orbital margin, which may be mistaken for the advancing border of acute dacryocystitis. The definite swelling or induration in the region of the sac with the nearly constant history of epiphora should suffice to make the diagnosis clear. The acute inflammation is treated on general lines—hot fomentations, attention to the bowels, etc. If pus forms, the abscess should be opened by free incision into the sac along a line passing downwards and outwards, commencing just below the internal tarsal ligament. Gauze plugging is then inserted. When the inflammation has subsided it will probably be found necessary to remove the sac.

The treatment of lachrymal fistula is similar to that of chronic dacryocystitis.

2. **Corneal ulcer.**—The pus in dacryocystitis contains a variety of pyogenic organisms, including the pneumococcus. Any slight trauma damaging the corneal epithelium is liable to be followed, in the presence of a purulent mucocele, by a virulent septic ulcer, often accompanied by hypopyon. This at the best will leave an opacity (nebula or leucoma) which seriously interferes with vision; at the worst, will necessitate excision of the eyeball. Ulceration is more likely to occur in elderly

DACTYLITIS

people in whose corneæ resistance to the pneumococcus is but slight. For treatment, see under CORNEA, AFFECTIONS OF, p. 291.

F. A. JULER.

DACTYLITIS.—Subacute or chronic osteomyelitis of a metacarpal or metatarsal bone or of a phalanx of the hand or foot; it may be due either to tuberculous infection or to congenital syphilis.

TUBERCULOUS DACTYLITIS

This form of dactylitis occurs in children and young adults, and may affect any of the long bones of the hand or foot.

Pathology.—The disease commences in cancellous tissue near the centre of the bone; the whole of the diaphysis becomes carious and the periosteum is inflamed; new bone is deposited on the surface, while the central part becomes caseous, so that pseudo-expansion of the bone results. Eventually the compact tissue is perforated and a subperiosteal abscess is formed; this may either spread through the skin and cause a sinus leading into the interior of the bone, or may spread to a neighbouring joint.

Symptomatology.—In infants several bones are usually infected at the same time, but in adults the disease is generally confined to one bone. One or more of the bones of the hand or foot is noticed to be swollen; pain is usually absent, but there may be slight aching; the muscles are wasted and the affected part is kept out of use as much as possible. The skin, as the inflammation approaches it, becomes hot and red, and a tender fluctuant swelling develops. If a sinus forms it is lined with pale granulations and the skin is undermined at its edges.

Treatment.—The general measures for treating all forms of tuberculosis must be employed, especially good food and fresh air. An affected finger must carefully be immobilized by the application of a splint, preferably poroplastic, including one of the neighbouring fingers and extending to the hand. In some cases it is possible to resect subperiosteally the whole of the diseased diaphysis. Failing this, the cold abscess must be treated by repeated aspirations, but if the skin is invaded it is necessary to incise it and to curette the bone cavity, removing any small sequestra. If possible, the wound should be closed without drainage, but it must be left open if there is secondary infection, being lightly packed with gauze soaked in paraffin

DACTYLITIS

containing 1 per cent. iodoform. For chronic sinuses, bismuth-paste injections may be used, but if the disease progresses and causes arthritis, amputation of the finger should be performed. In dactylitis of the toes amputation should be resorted to early, except in the case of the great toe.

SYPHILITIC DACTYLITIS

Syphilitic dactylitis results from congenital syphilis, and affects infants and young children. It is usually symmetrical, and most commonly occurs in the proximal phalanges.

Pathology.—There is sclerosis of the whole bone, with the formation of a central gumma; the periosteum is inflamed and deposits new bone, so that the phalanx becomes bulbous. The gumma eventually breaks through the peripheral bone, and, reaching the surface, leads to ulceration of the skin with discharge of gummy material; pyogenic bacteria then gain access to the bone, and necrosis, with the formation of sequestra, is produced.

Symptoms.—There is a painless, slowly progressive expansion of one or more of the phalanges of both hands; the overlying skin is oedematous, red and somewhat tender as the gumma approaches the surface. The disease produces a permanent shortening and thickening of the bone.

Diagnosis.—In the absence of associated lesions it may be impossible to distinguish syphilitic from tuberculous dactylitis by clinical appearances alone. The syphilitic variety is usually symmetrical and affects the proximal phalanges, and unless there is secondary infection it does not cause arthritis. The X-ray appearances are somewhat similar, but in tubercle there is as a rule great rarefaction, while in syphilis the density may be characteristic; in both forms small sequestra occur. A positive Wassermann reaction may be the only definite means of diagnosing syphilis.

Treatment.—The lesions usually clear up with treatment by mercury, but the early non-ulcerative stage may be very resistant; in such a case the bone must be opened with strict asepsis and its soft interior curetted; the resulting cavity is smeared with Bipp (bismuth 1, iodoform 1, paraffin 2), and the wound is closed without drainage. Curetting, sequestrectomy, and drainage are necessary where there is secondary septic infection.

Occasionally, subacute osteomyelitis of the metacarpus, metatarsus, or phalanges results from blood-stream infection with pyogenic bac-

DAY BLINDNESS

teria, especially in chronic pyæmia. The progress of the disease is more rapid than in syphilitic and tuberculous dactylitis, and there is more pain and tenderness. The cases must be treated by incision, opening the bone, and sequestrectomy in the later stages; a culture of the pus should be made, and a vaccine should be prepared from the organism which has been isolated.

C. W. GORDON BRYAN.

DANDRUFF (see SEBORRHOIC DERMATITIS).

DANDY FEVER (see DENGUE).

DAY BLINDNESS AND NIGHT BLINDNESS.

—The terms *nyctalopia* and *hemeralopia* were formerly used to denote night blindness and day blindness respectively, but different writers and nationalities used them in contradictory senses. Consequently these terms are better avoided. The conditions themselves arise mainly as a result of retinal or optic-nerve lesions, though one of the commonest causes of day blindness is cataract. Broadly speaking, any lesion which lowers the sensibility of the macular area of the retina causes day blindness, and lesions affecting the peripheral retina cause night blindness. In a normal person in full daylight the sensibility of the retina rises slowly from the periphery to the neighbourhood of the macula, and then very rapidly at the macula itself to a high peak, but in the dark-adapted eye the macular area becomes less sensitive than the area around it, so that the curve of retinal sensibility, instead of showing a peak, actually shows a depression in the centre. The consequence of this is that if there is any macular lesion the rise of macular vision does not take place in light-adaptation, and day blindness results, but in twilight vision approximates more closely to the normal.

When the peripheral field of vision is very restricted the patient may have good macular vision, but as the eye becomes dark-adapted the physiological depression of macular vision which takes place results in almost complete blindness. Apart from visual defect due to cataract and to opacities of the media, day blindness occurs in tobacco and other toxic amblyopias, in retrobulbar neuritis when there is a central scotoma, and in macular retina lesions. Night blindness occurs in retinitis pigmentosa, in secondary retinal atrophy resulting from disseminated choroiditis, in some cases of Leber's atrophy, and occasionally in aggravated cases of malnutrition. In the

DEAF-MUTISM

last-named cases the night blindness may be associated with xerosis conjunctivæ and may prove the precursor of kerato-malacia. High myopes often show some degree of night blindness. There is a rare form of congenital hereditary night blindness which has been traced in the case of one family through nineteen generations, and a still rarer form of congenital day blindness associated with complete colour blindness.

LESLIE PATON.

DEAF-MUTISM.—Children are seldom born deaf. Many, if not most, cases of deaf-mutism owe their disability to the onset of deafness between birth and the age of 4 years. The deafness being therefore usually acquired, we may believe that to a great extent it is preventable. Grave congenital deafness does occur, of course, and there is also an inherited type, as the existence of familial deaf-mutism clearly shows. The causes of such congenital and inherited deafness are not yet fully known. Some cases are due to developmental anomalies of the hearing apparatus; others to intra-uterine diseases like hydrocephalus, syphilis, etc.; but in the inherited variety the abnormality which is transmitted has not yet been discovered. Severe acquired deafness may be produced by cerebro-spinal meningitis, by mumps, and by such simple local conditions as suppuration or catarrh of the middle ear, labyrinthitis, etc.

Deaf-mutes are not necessarily also degenerates in other respects; they are, on the contrary, quite frequently persons of unusual sharpness of intellect.

Diagnosis.—Unless deaf-mutism is a known family trait, the suspicion that a child is seriously deaf does not dawn upon its parents, as a rule, until it is from 18 months to 2 years old. Then the fact that the infant is "backward in talking" attracts their attention, and it is at this time that medical aid is sought.

To ascertain whether or not a child is so deaf that it will probably become a deaf-mute is frequently very difficult, and may necessitate repeated examination. The difficulty is due to the fact that, if deaf, the child, even at this early age, has begun to develop its other senses to replace that of hearing. For this reason, the examiner must avoid making rapid movements which will attract the patient's visual attention, or producing vibrations that can be felt. Such acts will be responded to by the child, and a false impression that hearing is present may be created. The simplest

plan is to endeavour to awaken the child when it is asleep by sounding a whistle or a dinner-bell. But it must be remembered that such loud noises may be heard and yet the patient be quite deaf to vocal tones. That being so, caution should be practised until the investigation shows whether the child's attention can be attracted by the voice. It is important also to determine whether or not the child has succeeded in saying any of the simple nursery words or syllables. Backwardness in speech without deafness is common, but even backward children can command some amount of baby talk.

The detailed functional testing of the hearing must necessarily be postponed until the child is older. The examination should include a careful search for local conditions likely to cause deafness, such as adenoids and catarrh or suppuration of the middle ear.

Prognosis.—Children who become seriously deaf before the age of 4 generally grow up mute, losing the small vocabulary they may have already acquired. If the loss of hearing takes place between the ages of 4 and 7, a certain amount of speech will remain. After the seventh year the patient generally retains enough for ordinary purposes, and may even add to his vocabulary.

The prognosis in congenital cases is more favourable than in acquired cases. Spontaneous improvement occurs in the former not infrequently, and occasionally local treatment succeeds in enhancing the hearing power.

Treatment.—Attention has already been directed to the importance of the local treatment of middle-ear disease when it manifests itself before the age of 4. Many of the causes which lead to severe deafness at this period of life are, unfortunately, beyond the reach of remedies, but every effort should be made to arrest such diseases as catarrh and suppuration of the middle ear (see OTITIS MEDIA).

In recent years much effort has been expended in perfecting the methods of teaching the deaf-mute to speak and to read the lips, and with gratifying results. In all populous centres in Britain special schools now exist for the education of deaf children, and they are turned out able to speak and to understand what is said by others in the most surprising manner. These schools do not, as a rule, receive children before the age of 7. Children whose hearing power is so defective that they cannot benefit by instruction in ordinary

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schools and classes are eligible for the special schools.

Deaf-mutism and marriage.—If deaf-mutism is a familial trait in the patient's family, he will be liable to transmit the tendency. On the other hand, if he is the only member of his family to be deaf-mute, and especially if the defect can be clearly traced to an accidental cause such as cerebro-spinal meningitis or middle-ear suppuration, then he is no more likely to beget deaf-mute children than any other member of the community.

DAN M'KENZIE.

DEAFNESS (see EAR, EXAMINATION OF).

DECIDUOMA MALIGNUM (see CHORION-EPITHELIOMA).

DECOMPOSITION (see POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES).

DEFECTIVES, MENTAL (see MENTAL DEFICIENCY ACT, 1913).

DEFICIENCY DISEASES (see PATHOLOGY, CHEMICAL; BERIBERI; PELLAGRA; RICKETS; SCURVY).

DEGENERATION, REACTIONS OF (see ELECTRICAL REACTIONS).

DEGLUTITION, DIFFICULT (see SWALLOWING, DISTURBANCES OF).

DEGLUTITION, PNEUMONIA (see PNEUMONIA).

DELAYED CHLOROFORM POISONING (see VOMITING, POSTANÆSTHETIC).

DELHI BOIL (see ORIENTAL SORE).

DELIRIOUS MANIA, ACUTE (see CONFUSIONAL INSANITY).

DELIRIUM.—A transitory mental disorder characterized by confusion of thought, disturbances of perception and appreciation of the environment, and motor restlessness, and generally accompanied by illusions, hallucinations, and fleeting, vaguely defined delusions. It is a syndrome met with in a great number of different diseases and, although in its typical forms it presents a familiar and easily recognized clinical picture, it shades off by imperceptible gradations into conditions to which the term delirium is not generally applied. These are, on the one hand, the more prolonged confusional states met with in the exhaustion psychoses, and, on the other hand, certain varieties of stupor (q.v.).

Two main types of delirium may be differentiated, febrile and afebrile, although they differ rather in the circumstances of their origin than in the intrinsic character of the delirium itself. Delirium tremens and acute delirious mania (Bell's delirium), although they present the essential features of a delirium, have certain specific characters which entitle them to be treated as distinct entities, and they will not therefore be dealt with here (see ALCOHOLISM; CONFUSIONAL INSANITY).

Etiology.—Both febrile and afebrile delirium occur as episodes in the course of or subsequent to some other disease. Two principal etiological factors are concerned—(1) the severity of the primary disease, (2) the nature of the soil provided by the patient's constitution and previous history. Thus delirium may develop in certain neuropathic individuals or in alcoholics, in spite of the fact that the primary disease is running a mild course. Delirium also originates in children far more readily than in adults.

Febrile delirium occurs in the course of the exanthemata, particularly typhoid fever, and various other acute infections—pneumonia, acute rheumatism, influenza, septicæmia, etc. Afebrile delirium also may occur in the course of the various acute infections, but more frequently it makes its appearance in the subsequent stage of convalescence; it is also met with as a sequel to various exhausting conditions such as hæmorrhage, childbirth, and severe emotional stresses. Certain drugs, belladonna and cannabis indica for example, are capable of producing conditions of delirium, and delirious phases may also occur in the course of epilepsy and general paralysis.

Pathology.—Delirium is presumably due to the action upon the cortical elements of toxins generated directly or indirectly by the primary disease. No specific anatomical or histological changes can be described.

Symptomatology.—Febrile delirium may be preceded by premonitory symptoms such as insomnia, restlessness, or mental hebetude, but its development is generally rapid. Restlessness becomes pronounced, and an increasing mental confusion is apparent. The patient is unable to understand what is said to him, cannot appreciate what is going on around him, and begins to misinterpret the stimuli which fall upon his senses. Illusions and hallucinations develop. He mistakes the identities of the persons present, the objects in the room and the patterns on the wallpaper as

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perceived perhaps as terrifying or loathsome animals, and everyday sounds are interpreted in various fantastic ways. Hallucinations of all the senses may be present. Transitory and constantly changing delusions develop: the patient may believe that the house is on fire, or that he is about to be murdered. He becomes altogether impervious to his actual surroundings, and his actions are initiated solely by the illusions, hallucinations, and delusions which pass rapidly across his mind. He shouts in terror, tries to get under the bed, or attacks blindly the persons about him. The emotional state is most commonly one of apprehension or terror, though occasionally phases of exaltation are observed. Sleep is disturbed, and the administration of food is difficult. The physical signs present are naturally dependent mainly upon the nature of the primary disease. In severe cases the delirium may pass over into a condition of stupor, but in general, when the height has been reached, the symptoms gradually abate and the mental state slowly returns to normal.

The intensity of the delirium is commonly, but by no means invariably, proportional to the height of the temperature, and rises and falls with this. The full development described above is, of course, frequently not attained. In some cases the symptoms do not develop beyond a mild restlessness and confusion of thought, with perhaps a dreamlike misinterpretation of the environment.

Afebrile delirium has a symptomatology identical in its essential features with that of the febrile form. Its duration is short, ranging from a few hours to a few days. But in severe cases the physical disturbance is often profound, and a state of exhaustion or collapse may be rapidly produced.

Diagnosis.—The diagnosis of febrile delirium generally presents no difficulties, and the characteristic clinical picture developing in the course of an acute infection cannot well be mistaken for anything else. *Acute delirious mania* (Bell's delirium) is differentiated by the absence of any indication of an acute infection or other primary disease, the exceptional intensity of the delirium, and the rapid physical prostration.

In the case of afebrile delirium the history of a preceding acute infection, shock, or other of the causes mentioned above, the rapid development of the characteristic features of delirium, the short course, and the speedy onset of signs of exhaustion, are the decisive

features. In the early stages uncertainty may exist as to whether the case is developing into one of the more prolonged exhaustion psychoses (e.g. *confusional insanity*), but these generally develop much more slowly and gradually, and the more prolonged course soon disposes of any doubt.

Delirium tremens is differentiated by the history, the characteristic tremor, and other signs of alcoholism.

Prognosis.—The prognosis of febrile delirium is mainly dependent upon that of the underlying disorder. Generally in itself it is not of great import except in those cases where it is very intense and prolonged, and the danger of exhaustion and collapse arises. The prognosis of afebrile delirium is usually good; unless the physical exhaustion is very intense, most cases recover rapidly. In a few cases some general mental weakness may persist for a considerable time.

Treatment.—The treatment of febrile delirium practically resolves itself into the treatment of the primary disease, and the delirium itself rarely requires other measures than precautions against the patient injuring himself in his excitement, and the preservation of his strength by suitable nourishment. The delirium is commonly proportional to the height of the temperature, and hydrotherapy or other means employed to lower the latter also reduce the former.

In afebrile delirium the chief indication is to maintain the patient's strength. This is secured by the administration of adequate nourishment and the adoption of sedative measures. Food must naturally be given mainly in liquid form, and at frequent intervals. Sedative measures consist in the employment of hydrotherapy and drugs. Prolonged warm baths (90°–95° F.) or warm wet packs are valuable, but must be used with caution if exhaustion and collapse are feared. Paraldehyde, sulphonal, or trional may be given, or, if the excitement is pronounced and it is difficult to administer drugs by the mouth, hyoscine hypodermically. Scopolamine and morphia combined have also been recommended. The free administration of liquids is generally advised in order to help in the elimination of the toxin presumed to be the cause of the condition, and attention should be directed to the free action of the bowels and skin. The action of the skin is also aided by the diaphoretic effect of hydrotherapeutic measures.

BERNARD HART.

DELUSIONS

DELIRIUM TREMENS (*see* ALCOHOLISM).

DELUSIONS.—A delusion may be defined as a false belief whose falsity would be obvious to a person of the same race, age, class, and education as the person who expresses it. The qualification contained in the latter part of the definition is necessary because merely false beliefs occur everywhere, and are not to be classed as delusions unless this further condition is satisfied.

A delusion is impervious to argument and demonstration, and, however convincing and weighty the opposing evidence may be, the patient's belief remains absolutely unshaken. Persistence in the face of destructive evidence is, of course, frequently observed also in normal beliefs, notably in the spheres of religion and politics, but the immutability here is relatively less than in the case of delusions proper, and can be broken down by the production of sufficiently conclusive evidence. The distinction between non-rational beliefs and delusions, nevertheless, is to be regarded psychologically as one of degree only.

Delusions may concern all possible subjects. The great majority, however, may be grouped under the three headings of grandiose, persecutory, and altered physical or mental personality. The patient may, for example, believe that he is the King, or that there is a conspiracy to deprive him of his liberty, or that his stomach has been removed. Delusions are also described as systematized or unsystematized according to the extent to which they are knit together into a coherent system, or isolated and without apparent relation to one another. They are often associated with hallucinations, e.g. the patient who believes himself to be a king may hear a voice addressing him by his regal title. It is often said that hallucinations may be the cause of delusions. Possibly this may be true in some cases, but generally the hallucinations and the delusions are to be regarded as different aspects of one and the same morbid process. The type of delusion present is commonly, though not invariably, in harmony with the dominating emotional state. Thus a melancholic is likely to exhibit depressive delusions, e.g. that he is eternally damned, while an exalted general paralytic is likely to exhibit expansive delusions, e.g. that he is the strongest man in the world.

In the sense in which they have been defined above, delusions practically occur only in the

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psychoses (insanities). They are met with in many different types of insanity, but it must be clearly understood that they rarely serve to mark off one variety from another, and that in many of the psychoses delusions are only a secondary feature, and may be absent altogether. The law, however, lays great stress upon their existence as evidence of insanity, and in drawing up medical certificates and other legal documents any delusions which can be elicited should always be mentioned.

One of the chief practical points to be ascertained with regard to a delusion is the extent to which it influences the patient's actions. In some cases conduct is apparently entirely unaffected, so that a patient who believes himself to possess untold wealth will contentedly spend his days scrubbing the ward floor. In other cases conduct is completely dominated by the false beliefs present, and in such circumstances a patient with persecutory delusions may become extremely dangerous. He may, for example, attempt to murder the person whom he believes to be persecuting him. This point is therefore of the first importance in deciding whether and to what extent the patient must be restrained and controlled. (*See* PARANOIA.)

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DEMENTIA.—Acquired enfeeblement of the mind is called dementia. The term should be restricted to this condition. In general literature it is often used loosely. For example, the expression "behaving like one demented" may mean showing signs of excitement or depression. In the literature of insanity dementia has had a place for many years, and in older works was divided into two main classes, primary, and secondary or chronic. The descriptions of primary dementia correspond in some respects to dementia præcox, and in others to stupor, so that "primary dementia" is gradually falling into disuse.

Varieties.—The varieties of dementia are (1) secondary, (2) alcoholic, (3) epileptic, (4) organic, (5) senile. Dementia is also the terminal condition of dementia præcox (*see* the next article) and general paralysis of the insane.

Etiology and morbid anatomy.—The causes giving rise to this condition will be mentioned when discussing the varieties, but it is generally believed that in most cases, though an extrinsic factor may appear to have a predominant influence, the essential cause is intrinsic—that is to say, a *fixed* staying power of the neurones hereditary

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transmitted. For example, in alcoholic dementia the cause is given as alcohol, though, as Maudsley said many years ago, certain men can drink themselves into their graves before they will drink themselves insane.

The conditions found post mortem are a general atrophy of the brain, shown by thin convolutions, large sulci, thickening of the pia-arachnoid membrane, and excess of fluid in the lateral ventricles. The ependyma of the ventricles is frequently granular, especially that of the lateral recesses of the fourth ventricle. The microscope reveals loss and degeneration of the neurones and increase in the supporting connective tissue.

Symptomatology.—The dementia may be so slight as to be scarcely noticeable, or so profound as to leave few signs of mind in the patient. In a case of medium severity the expression is vacant, and changes but little, and then only by a fatuous smile. The intellect, the emotions, and the will-power show signs of deterioration. Evidence of this will be obtained most easily by an examination of the memory, which as a rule is most defective for recent events, while some old memories may remain. Recent acquisitions, such as languages and accomplishments, go first. The edge of the emotions, whether sad or joyful, is dulled, and initiative is at a standstill. Many of these patients can be trained to be excellent workers, but when the motive power supplied by the watchful care of the nurse is withdrawn they sink into listless idleness. If delusions or hallucinations have been present in the initial illness they often persist, but have little influence on conduct. In the final and severe cases the mental condition may be that of a baby. The patient may be dirty in his habits and make no attempt to control the bladder or rectum. He may have to be washed, dressed, and fed. In addition, he will probably have degraded habits of all sorts, being destructive and without shame, and an occasional meaningless outburst of violence may take place. There are no special physical signs. In mild cases cyanosis of the extremities and oedema of the feet and ankles are often present. In severe cases the patient may curl himself into rigid attitudes which may lead to contractures. A special odour used to be thought to emanate from the insane. The skin is greasy in many cases, and the sweat appears to be excessive, but if ordinary cleanliness is insisted upon no special smell is apparent.

Secondary or sequential dementia.—This form follows a severe mental illness, most commonly mania or melancholia. In these cases symptoms of the preceding affection colour the symptoms of dementia, but they have lost their acute character.

Alcoholic (see ALCOHOLISM.)

Epileptic.—The terminal condition of many cases of epilepsy is dementia. This is often attributed, on insufficient evidence, to the bromide which has been given. These patients are apt to be quarrelsome and mischievous, and to have occasional outbursts of extreme excitement. Many also show a tendency to excessive religious display.

Organic.—This term is applied to those cases in which dementia has been caused by a gross brain lesion, as hæmorrhage, softening, tumour, etc.

Senile (see DEMENTIA, SENILE).

Diagnosis.—The general diagnosis, as a rule, presents little difficulty. As regards differential diagnosis, the conditions which most resemble dementia are congenital defects, dementia præcox, stupor, and general paralysis of the insane. From *idiocy* the diagnosis can often only be made from the history. Even in the last stages of *dementia præcox* some remains of the initial stages of the illness, such as catatonia or mannerisms, are apparent, though in a feeble form. *Stupor* is a temporary abeyance of the mental functions and is often indistinguishable from dementia. Time alone will tell if the condition is permanent. If the dementia is that of *general paralysis of the insane* the physical signs in the pupils, speech, and reflexes, and an examination of the cerebro-spinal fluid, can remove all doubts.

Prognosis.—This is hopeless as regards mental recovery. As regards life, with care mild cases of dementia may survive for years. Death is usually caused by intercurrent disease, and the patients fall an easy prey to tuberculosis or dysentery.

Treatment.—The great aim in the treatment of the insane is to prevent dementia. When failure has to be admitted, then the aim is to prevent the development of a severe degree of the condition. There is no doubt that persistent attention to the patient and never-wearying attempts to prevent his falling into a condition of listless carelessness will work wonders. It must be remembered, too, that numbers of neurones remain which can be educated to take the place of those that have been destroyed. Many of the

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patients, therefore, can be trained to useful work, and thus mitigate the economic loss sustained by the community which supports them. The condition as regards dress, general appearance, and behaviour of the demented in an asylum often forms a useful index of the management of the institution.

R. H. STEEN.

DEMENTIA, ALOOHOLIC (*see* ALCOHOLISM).

DEMENTIA, CATATONIC (*see* DEMENTIA PRÆCOX).

DEMENTIA, EPILEPTIC (*see* EPILEPTIC INSANITY).

DEMENTIA, HEBEPHRENIC (*see* DEMENTIA PRÆCOX).

DEMENTIA PARALYTICA (*see* GENERAL PARALYSIS OF THE INSANE).

DEMENTIA PARANOIDES (*see* DEMENTIA PRÆCOX).

DEMENTIA PRÆCOX. — A psychosis, becoming evident most frequently in early adult life, characterized by varying clinical symptoms and profound disturbances of personality, judgment, and will, and tending in nearly all cases towards progressive and often rapid dementia. The forms in which dementia præcox may be observed have been described under other names and as distinct diseases—as catatonia, a form of melancholia, primary dementia (*see* DEMENTIA), or as allied to general paralysis, while paranoid dementia was classed with true paranoia (q.v.).

The epoch-making work of Kraepelin consolidated the conception of the disease as a whole and served to show the fundamental relationship between forms which differed widely in early symptoms.

Etiology.—The disease arises most frequently in families already tainted with insanity, epilepsy, or other neuroses, but isolated cases may occur in which no insane heredity can be traced. Clouston speaks of the insanity of adolescence and of puberty as “the most hereditary of all forms of mental disease,” and various authorities have recorded that a family history of insanity is found in from 50 to 90 per cent. of cases. Not infrequently cases have been recorded of brothers and sisters suffering from it, and eccentric characters, criminals, and others showing mental or moral defects, may be found among the

near relations. The incidence is about equal in both sexes, the hebephrenic form being more common in men, the catatonic and paranoid forms in women. Cases of dementia præcox probably form about 12 per cent. of all admissions to institutions for the insane. The majority of cases develop between the fifteenth and thirtieth years, but a few may be found in the fourth and fifth decades.

Inquiry as to the personal history of patients suffering from this disease shows in a large proportion of cases that peculiarities of character have been noticed from an early age; that the patients have been “not like other children,” very quiet and seclusive, or abnormally excitable and unmanageable, dreamy, and unpractical.

The frequency with which the onset occurs about the period of puberty and adolescence, during pregnancy, or subsequent to childbirth, and the fact that disturbances of menstruation are frequent, have lent support to a theory that the disease is due to faulty metabolism of the sexual glands. This is confirmed by the researches of Mott and others, who have demonstrated a regressive atrophy of the testes with, in a large proportion of cases, complete arrest of spermatogenesis; the ovaries also show degenerative changes.

Symptomatology.—The outstanding feature of dementia præcox is affective indifference and apathy, the conscious self appearing to lose its intimate relation to the patient's surroundings. The further the disease advances, the more he lives in a world of his own: his reaction to normal stimuli resembles that of a sleep-walker who acts mechanically, though his conscious will plays no part in the action. Interest in ordinary affairs and in the surroundings is lost to a great extent, and there is a deep disturbance of the will-power; a disconnection between thought and action, with extreme emotional indifference. Patients laugh, cry, or have violent outbreaks of anger without any adequate cause; they lack initiative and persistence in the ordinary business of life, and sink easily into dependent positions; they may profess themselves in love with some real or imaginary individual, and weave romances, but all in an unreal and vague way.

The onset of the disease may be so slow and gradual that no suspicion of insanity is entertained at first by the patient's friends and the symptoms are regarded as slight eccentricities, or defects of character, rather

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than as mental disease. On the other hand, an attack may come on suddenly in a patient who has previously been in good health. Negativism, one of the most constant symptoms, may show itself first as a tendency to argue, contradict, and act in opposition to all advice. Later, as it becomes more marked, the mere fact of any action being suggested is enough to make the patient take an opposite course. Automatic obedience often appears with negativism, and yet in curious contrast to it. Patients who mechanically resist everything that is suggested or done for them may settle down with hardly a protest to the life of an institution, and the marked defect in will-power shows itself in a lack of initiative and tendency to act in stereotyped grooves.

Physically, patients are often of the thin, weedy type, narrow-chested and poorly developed, with simian hands and feet, deformed ears, or other stigmata of degeneration. Most of them suffer from defective circulation, low blood-pressure, cold clammy and cyanosed hands and feet, chilblains, etc. In the acute stages, exaggerated knee-jerks are often present, and headache (especially vertical and occipital), insomnia, and constipation are common.

The way of shaking hands is very characteristic; instead of grasping, the hand is held out limply and takes only a passive part in the act.

The seclusiveness which is often an early symptom may be associated with masturbation, and the latter has been assigned as a cause of the insanity, but it should rather be regarded as a consequence of the mental deterioration.

Hebephrenic type.—Patients become restless, excited, talkative, their powers of concentration are poor, and they do not attend to any occupation or interest for long. As the attack advances, they become silly in behaviour, speech is disconnected or quite incoherent, they make grimaces, laugh or cry for no adequate reason, and become untidy in dress. Delusions, usually of a fleeting character, are present, and patients will romance, and relate experiences with no foundation in fact.

Auditory, olfactory, or visual hallucinations are often present, and the patient laughs, talks, or becomes angry in answer to "voices." In the more excited cases, sleep is broken, and for weeks or months there may not be more than an hour or two each night; and the motor restlessness is extreme both

day and night. Patients react badly to sedatives and become very thin; the expression is haggard, the skin muddy, and the hair rough and lustreless. They may be too restless to eat, throwing food about or upsetting it; or, on the other hand, may eat voraciously. They may become destructive and dirty in habits.

After this acute stage, there may be a remission, indeed apparent or temporary recovery, or else as the severe symptoms subside the patient becomes quieter, fatter, more indifferent, and sinks gradually into a terminal dementia.

Catatonic type.—In this form the signs of negativism and stereotypism are very marked. There is an apparent lack of interest in surroundings, reluctance to answer any questions or respond to external stimuli, or there may be complete mutism, the subject remaining absolutely silent for weeks or months at a time. That this indifference is not the result of mental confusion is shown by the fact that later such a patient may remember facts that occurred during the mute stage. The disturbance of will-power is shown both by negativism and by automatic obedience. For example, a patient sitting rigid and irresponsive may resist strenuously an effort to raise his arm, yet, when the movement has been accomplished in spite of him, the arm will remain in the raised position for some minutes—the so-called "*flexibilitas cerea*." Echolalia or echopraxia may also be present, the patient repeating any word or action which is rehearsed before him. Stereotyped movements and repetition of certain words or sentences are commonly noted. Patients stand rocking from one foot to another, make inarticulate humming noises, and stroke or rub their faces. The same movements may persist for many years, even after an advanced stage of dementia has been reached, or they may occur as a transient symptom of an attack which shows varying phases and remissions. Sitting or lying in strained and uncomfortable positions is common; especially characteristic are those of lying on the back with the head raised though unsupported, or sitting bolt upright with a hand laid flat on each knee. Mutism commonly accompanies other forms of resistiveness, as refusing food and objecting to and resisting dressing or undressing. Voluntary retention of fæces is sometimes a troublesome symptom, and saliva may be retained in the mouth for hours together. Impulsive

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violence is common: an apparently lethargic and apathetic patient will spring up suddenly and strike someone for no reason.

The patient may either gradually improve and make an apparent recovery, or the same symptoms may persist till dementia supervenes, or one of the other types, as the hebephrenic, may take the place of the catatonic form.

Paranoid type.—This shows a marked resemblance to true paranoia, and includes many cases which were formerly grouped under that name. It is usually of slow onset, and frequently occurs in patients who have always been inclined to solitary habits, unsociable and suspicious, and disposed to brood over small injuries and imagined slights. Gradually the subject becomes more wrapped up in himself and depressed, and begins to suspect those around him of plotting against him, or to think that he is the subject of mysterious and unseen influences from someone at a distance. He hears "voices," which tell him of persecution directed against himself, or speak to him by telephone or telepathy. Female patients may believe that they are being outraged, and that men enter their rooms at night. Often they mention love affairs with unknown or imaginary persons, and, even if there is a real object, the affair often has an unreal aspect. Outbreaks of excitement and violence often occur, and sometimes extreme depression may lead to suicidal impulses.

Simple demented type.—In some cases patients who have shown the premonitory signs of dementia præcox—seclusiveness, affective indifference, and lack of initiative—become progressively weak-minded, and sink more or less rapidly into dementia without exhibiting any of the more striking symptoms peculiar to the types already described. Many cases remain as partial demented without degenerating into the more pronounced forms. To this class must also be added those who, apparently normal or even brilliant in youth, show a tendency to premature degeneration of intellectual powers, and an inability to stand the strain of prolonged mental work. The student who, though carrying everything before him in his school and college career, yet proves himself mediocre and unequal to the responsibility of his actual life's work, must be regarded as an instance of premature dementia, though not insane in the ordinary sense. In some patients incoherence of speech, both written and spoken, is very noticeable early in the case

and before the dementia is very advanced, but such cases do not differ materially from others, and may show vague delusions and other symptoms of the more classical types.

Moral type.—In a certain number of cases the earliest and most noticeable symptom is a degeneration of the moral character, usually followed later by other characteristic features of the disease. Haury cites a case in which a young soldier who was suspected of malinger and simulating defective vision was punished for extreme inertia at drill. He appeared to simulate mental defects, then developed negativism and stereotypy, refused food, etc., and, on being seen by an alienist, was shown to be suffering from the catatonic form of dementia præcox.

Circular type.—Some patients show a periodic variation in symptoms, becoming excited or depressed by turns; in the early stages these cases may resemble those of true circular insanity.

Diagnosis. *From mania.*—The hebephrenic type may closely resemble mania, but the attacks tend to last longer, speech is more incoherent even when the acute excitement is past, and there is not the rapid reaction to external stimuli which is shown by the maniac. As a rule there is a history of a gradual onset of mental symptoms, peculiarities of character, mannerisms, or seclusiveness, existing from an early age.

From melancholia.—The catatonic variety is that most likely to be mistaken for melancholia, but careful observation will usually reveal some distinctive features, such as automatic obedience, stereotyped movements or attitudes; and while the melancholic is ready and willing to pour out his troubles to anyone who will listen to him, the subject of dementia præcox is reserved, expresses his sorrow more vaguely, and may even show complete mutism.

From paranoia.—The paranoid variety closely resembles true paranoia, but hallucinations, especially of hearing, occur more constantly than in the latter disease; there is less systematization of the delusions, and dementia occurs earlier.

From epilepsy.—There may be symptom resembling petit mal, when patients appear momentarily unconscious of their surrounding and indifferent to questions or to touch. These are due, however, to "voices" which for the moment completely absorb their attention.

From general paralysis of the insane.—A case of dementia præcox with exaltation may sup

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ficially resemble general paralysis of the insane, but a careful physical examination will probably make the difference clear, and, should there be any doubt, the Wassermann test of the cerebro-spinal fluid will settle the question.

From circular insanity.—Careful observation will usually discover in the subject of dementia præcox an emotional indifference and negativism and a tendency to early dementia that are not found in circular insanity.

Pathology.—Alzheimer and other investigators have observed various changes in the cerebral cortex of patients suffering from dementia præcox. The normal arrangement of the nerve-cells is altered, they appear swollen, rounded, or atrophied and stain badly, and their nuclei are enlarged, eccentric, or extruded. The fine axis-cylinders degenerate and there is an increase of neuroglial tissue. The changes are most marked in the frontal region and, especially in catatonic cases, in the deeper layers of the cortex. There is no perivascular infiltration or vascular proliferation.

Prophylaxis and treatment.—In children predisposed by heredity, every effort should be made to secure sound physical health: they should lead a healthy outdoor life, and education should begin late and be without ambitious aim. Clouston lays stress on the desirability of neurotic children taking a diet from which meat is to a great extent, and alcohol entirely, excluded, and which contains plenty of milk and fats.

When the disease has actually developed, institutional treatment is nearly always necessary—the patient may have suicidal tendencies, acute excitement, or restlessness, and cannot be treated adequately at home. In many cases where the general health is poor, rest in bed is required, if possible in the open air, with attention to adequate sleep and food. Should there be much excitement, with loss of sleep, the continuous warm bath should be tried, careful watch being kept on the pulse. The bath should be given at first for half an hour, at a temperature of 98° F., before bedtime. Sometimes patients who are too restless to be kept in the bath will tolerate it after a few doses of a sedative such as sulphonol. Plenty of nourishing food is of great importance, especially in the thin, anæmic type of case. Extra milk, eggs, fruit, etc., should be added to the ordinary diet, and there should be no hesitation in feeding by tube should the patient refuse food. If there is much

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tendency to violence and self-injury, a padded room may be necessary.

With regard to drug treatment, the sluggish circulation and low blood-pressure so often found in this class of patient should be stimulated by nux vomica, digitalis, etc. For this purpose, Rae Gibson has advocated supplementing the drug treatment by cold baths, exercises, etc., and has reported good results. Constipation should be constantly attended to and controlled. For insomnia, sulphonol should be used sparingly, and never for more than three or four nights in succession without an equal interval and a dose of calomel, owing to the danger of hæmatoporphyrinuria from its cumulative effects. Adalin is safer, and gives good sleep without any unpleasant after-effects. Prescribed in doses of 15 gr. at bedtime, it often gives a restful night; it sometimes acts better if 5 gr. are given about five o'clock and 10 gr. at bedtime. Paraldehyde is safe, acts quickly, and can be given for long periods if necessary. Its objectionable taste can be disguised to some extent by tincture of orange.

With each individual the reaction to drugs varies, and the right one must be found, and, of course, none given where other therapeutic measures will answer. E. M. JOHNSTONE.

DEMENTIA, SENILE.—A certain amount of emotional apathy, failure to form new thought-associations, and lack of initiative is physiological in old age. When this is extreme in degree it is pathological and is known as senile dementia, or presbyophrenia.

Etiology.—Heredity plays a large part in the causation; a tendency to premature senility is met with in certain families. Syphilis, lead poisoning, and the abuse of alcohol cause arterio-sclerosis, a condition frequently met with in senile dementia. Other causes, such as the stress and worry of an anxious and overworked life and the abuse of tobacco, are of doubtful etiological importance.

Pathology.—The brain is atrophied, especially in the frontal regions. The pia-arachnoid is thickened and full of fluid. The convolutions are thin and worm-like, and the sulci large. Patches of softening may be found in various places. The ependyma, especially that of the lateral recesses of the fourth ventricle, is granular. Under the microscope the number of neurones is found to be decreased, and many of those that remain are atrophied. Senile changes coexist in the viscera, and interstitial nephritis is often present.

Symptomatology.—The mental symptoms differ but little from those of other forms of dementia (*see* DEMENTIA). The intellect is generally enfeebled, the emotions are dulled, and the will-power almost gone. Of the intellectual faculties, the memory is the easiest to examine. Anterograde amnesia (loss of memory for recent events) is marked, and it is astonishing to find the memories of bygone years persisting in normal fashion when the events of yesterday are forgotten. The judgment is unreliable, and in mild cases a frequent feature is the magnification of small worries into great catastrophes. As regards the emotions, while severe losses may be borne with equanimity, the patient often weeps for little reason. The habits are, as a rule, faulty, and all sense of modesty is lost. In severe cases the patient leaves the world in a condition not dissimilar from that in which he enters it, requiring to be washed, fed, clothed, and generally tended like an infant. A common and difficult feature is the extreme restlessness and press of activity found in many cases. In the middle of the night the patient will insist that it is morning, that he must be up attending to his business, and he will rise, forgetful of his feebleness, and struggle with his nurses.

The physical signs are mainly those of the cardio-vascular lesions usually associated with senility, viz. thickened arteries, feeble irregular heart, high blood-pressure, and signs of granular kidney.

Diagnosis.—This is usually a matter of little difficulty. Senile dementia associated with premature senility may appear at a comparatively early age, and, as *general paralysis* may occur at a similar age, this disease must be excluded before a final diagnosis is made. Moreover, the tremor of senility may be confused with the tremor of general paralysis. In the latter disease the ocular signs, the speech, the condition of the cerebro-spinal fluid, and the other physical signs will render diagnosis easy. Thickened arteries are not present in all cases of senile dementia; some authors describe a separate variety under the heading arterio-sclerotic insanity. In the latter, signs of focal lesions (softening) may be present, but otherwise there is little difference from the condition under consideration.

Prognosis.—The disease progresses to its termination by death, but often the downward movement is one of unexpected slowness, life being prolonged for many years. Unless the

patient is carefully protected there is the danger of attack by intercurrent diseases, such as pneumonia.

Treatment.—The question whether the patient should be nursed at home or sent to an institution is often a difficult one to determine. There is the natural sentiment that if the patient is going to die he should do so at home, but in severe cases he is best cared for in a mental hospital. The restlessness referred to above makes the case an anxious one for the nurse. There is always the danger of a fall with a broken limb or rib, and a padded room is of great assistance in this respect. On the other hand, there are mild, quiet cases with little mental enfeeblement which ought not to be sent to mental hospitals. It is not pleasant for young people to confess that their grandfather died in an asylum.

The treatment of a case of senile dementia resolves itself largely into a question of nursing. The diet must be of milk and slops, and careful watch must be maintained against choking through the patient bolting his food. The skin must be protected against bedsores, and this is not an easy matter owing to the dirty habits. Constant cleansing when wet is the only remedy, and it is as well to keep the patient up for as long a time as possible daily. In males with enlargement of the prostate the catheter may have to be used. For the restlessness bromides may be of value, and paraldehyde is the best hypnotic.

R. H. STEEN.

DENGUE (*syn.* Dandy Fever, Breakbone Fever, Stiffnecked Fever, Bucket (bouquet) Fever, Polka Fever (Brazil).—An acute infectious fever of the tropics and subtropics usually occurring in epidemic form.

Geographical distribution and epidemiology.—Dengue exists in epidemic form in most parts of the world, especially along the coasts and main routes of travel, though in the hot weather it has occurred outside these zones, as, for instance, in Philadelphia, Boston, and New York. Many epidemics have been described. The earliest record is that of 1779 in Java and in Egypt. The disease is always endemic in the Pacific islands.

Dengue attacks all races and sexes indiscriminately, but recent arrivals are more susceptible than older residents. It appears to have a definite seasonal incidence, and the tropics occurs after the rainy season; instance, in the Pacific islands in the month of June, July, and August. It may attack

DENGUE

large proportion of the population ; thus 20,000 people were affected in Galveston within a period of two months in 1897.

Etiology.—Various organisms have been described in dengue, but so far none has been authenticated. In 1916 Cleland and Bradley in Queensland initiated a series of experiments on a strictly scientific basis, and concluded that *Stegomyia fasciata* is the chief carrier of the germ. They also succeeded in proving that the virus exists in the blood from the second to the fourteenth day of the disease.

In 1917 Koiyumi, Yamaguchi, and Tonomura, in the Formosan outbreak, incriminated other mosquitoes allied to *S. fasciata*, such as *S. scutellaris* and *Desvoidia obturbans*; they also claim to have obtained positive results with *Culex fatigans* in support of earlier work of Graham's. These Japanese writers further proved that, such is the concentration of the virus, that 0.00005 c.c. of dengue serum, when injected intravenously, is capable of reproducing the disease in a non-immune.

In contrast to yellow fever, the incubation period of the disease in the body of the mosquito host is unknown.

Pathology.—As dengue is not usually a fatal disease, its true pathology is not accurately known. A report of the fatal cases in 1885 in Galveston, Texas, records localized inflammations of serous membranes, especially the pleura and peritoneum. Hirsch mentions serous effusions in the neighbourhood of joints and inflammation of the crucial ligament of the knee.

Symptomatology.—The incubation period is stated by Australian workers to be from five to nine days, during which time the patient experiences slight headache. The Japanese, more precise, give the period as 130 hours. The course of the disease can be divided into three periods—those of (a) invasion, lasting 48–72 hours, (b) remission, lasting 12–72 hours, (c) the return of early symptoms, lasting 24–36 hours.

Stage of invasion.—In the majority of cases the onset is sudden; it may come on during sleep, the patient awaking startled with the pain, but this varies greatly with the severity of the attack. This stage is accompanied by malaise, complete anorexia, fever, headache, and slight shivering. The prostration may be severe in adults and even worse in children, and delirium may occur. The pains are often so intense that all the bones feel as if broken, a sensation which makes the victim maintain a

rigid attitude. They may affect any portion of the body, but especially the knees; there is a noticeable absence of swelling and of tenderness on pressure over the joint. The temperature may rise to 106° F., falling after three days as suddenly as it rose, often with sweating. The pulse is slow in comparison with the pyrexia, a rate of 75–90 being recorded with a temperature of 102° F. (Goldsmid and Crosse). The tongue is generally covered with a thick white central fur, but is clean at the sides.

Stage of remission.—Directly after the subsidence of the fever the patient feels better and gets up, but has still a dry tongue and general feeling of stiffness.

Third stage.—After three days the primary symptoms may return, perhaps with increased violence; the temperature rises again and is accompanied, in 40 per cent. of cases, by an eruption. This stage may be absent.

Convalescence is generally prolonged; there is considerable weakness, and residual pains cause a good deal of disability. The most striking feature is bradycardia. I have seen cases with a persistent pulse-rate of 44–48. The crippling that results has probably given the disease its name of "dandy fever."

The **facies** is generally stated to be characteristic; there is a general suffusion with injection of the conjunctiva and tumefaction of the lids, but without lachrymation or coryza, so that the patient resembles a man recovering from an alcoholic bout.

Pains.—The pains of dengue have been described as "the earliest harbinger, the persistent companion, and the last vestige of the disease." The general body pains have been compared to the soreness felt after being beaten with sticks. Restlessness and inability to remain long in one position are characteristic. Pain in the back of the neck and in the lumbosacral region is common. General aching of the limbs seems to be responsible for the pains in the knees rather than involvement of the joints themselves; it occurs in 69 per cent. of cases. Headache is recorded in 93 per cent. of cases, but the intensity varies very much, and the aching does not appear to be located in any particular area. Pain in the eyeballs is present in 27 per cent. of cases; it is apparently situated in or at the back of the eyes; cases have been recorded in which the patient turned his head rather than attempt to move the extremely sensitive eyes. Abdominal pains may exist with diarrhoea.

Mental symptoms consist of mental irrita-

DENGUE

bility, and a very characteristic depression lasting well on into convalescence. Insomnia is frequent, and may complicate convalescence. Delirium is seldom present.

Gastro-intestinal symptoms.—The dirty tongue, furred at the back, with "strawberry" tip, has been compared to that of scarlet fever; nausea without vomiting is fairly common; diarrhoea may be present.

Enlargement of the lymphatic glands has been described by several observers; the glands are said to be tender.

Urine.—The specific gravity is normal and the reaction acid; most observers record the absence of albuminuria.

Blood.—There is a definite *leucopenia*, the white cells falling sometimes as low as 2,000, with a relative increase in the large lymphocytes; this occurs about the fourth day of the disease. As low a count as 1,200 has been recorded by Ashburn and Craig.

The **skin eruption** is generally described as being of two kinds—prodromal and terminal. The *prodromal* rash is a general erythema, "a fine punctiform rash, usually found over points of friction" (Goldsmid and Crosse). There are hypersensitiveness of the skin and *tâche cérébrale*. The *terminal eruption* is usually described as being rubeoloid, but there is great diversity of opinion. It may be present forty-eight hours from the onset, may not be noticed till the fifth day of the disease, or may be absent altogether. It generally occurs as dark dusky spots 2-3 in. in diameter with healthy intervening skin, affecting the palms of the hands and extending up the arms to the trunk, thigh, and legs. It is devoid of special features, and is said to be "midway between scarlet fever and measles, but less definite." Together with the rash there is often a reddening of the elbow. Desquamation of a fine branny character succeeds, and is accompanied by intense itching, which may last for two or three weeks.

Diagnosis.—From *measles* the differentiation is made by the absence of catarrhal symptoms and Koplik's spots; from *scarlet fever*, by the absence of angina, quick pulse, and leucocytosis. *Typhus* often presents a difficulty. The more serious mental condition of the typhus patient, the high remittent temperature, the petechial character of the rash, and the Felix-Weil reaction, help in diagnosis. Dengue and *yellow fever* have several points in common, such as leucopenia and slow pulse. The absence of rash, the jaundice, and the

DENTAL CARIES

albuminuria in the latter should be borne in mind. From *phlebotomus fever* dengue is distinguished by the rash and the absence of conjunctival symptoms, though this is by no means always easy; some indeed declare the two diseases to be identical. Dengue differs from *influenza* in the more intense character of the bone pains and the absence of respiratory symptoms. *Enteric* often resembles it closely at first, but the bone pains and remittent character of the temperature and the serum reactions should assist. Dengue is liable to be confused with *rheumatic fever*, but is unaccompanied by joint-effusions and profuse sweats.

Complications are few, and consist of insomnia, delirium, debility, and delayed convalescence. Relapses tend to occur for as long as two weeks after the original attack.

Treatment is entirely symptomatic. The bowels should be well opened with calomel and saline. It is useless to attempt by means of drugs to cut the pyrexia short; one cannot prevent the disease from running its natural course. For the headache, phenacetin or aspirin is given. For the joint-pains, salicylates alone or in combination with an opiate are useful. If the pains are very severe an injection of morphia may be required. A pill composed of phenacetin 5 gr., codeine $\frac{1}{4}$ gr., and camphor monobromate 1 gr. can be recommended. The diet should be light, and for the debility of convalescence a good red wine is very helpful.

P. MANSON-BAHR.

DENTAL CARIES.—This is probably the commonest disease occurring among civilized races; among the uncivilized it is comparatively rare.

An affected tooth is gradually destroyed if the caries is allowed to progress undisturbed. Even rapid caries continues for several months before the crown of the tooth is extensively destroyed, but when a large cavity has broken down the destruction is much delayed, the conditions being less favourable to its progress. When the crown is lost, dental caries makes exceedingly slow progress in the root, which may remain *in situ* for several years if undisturbed. Infection and loss of function lead to absorption of the tooth and of the supporting tissues, so that in due course the root lies on the surface, attached to the gum by fibrous tissue.

The disease progresses in two stages, (1) by decalcification, (2) by digestion of the organic matrix. Decalcification is due to the action

DENTAL CARIES

of organisms upon carbohydrates and the production of organic acids, chiefly lactic acid, which remove the lime salts. Subsequently organic matter is acted upon by organisms, either directly or through ferments which they produce.

The enamel is first attacked at a point where food lodges, the two chief sites being the fissures, particularly those situated on the crowns of the molars and premolars, and the points of contact between adjoining teeth. The first permanent molars are remarkably prone to caries; they erupt at the age of about 6½, and the flap of gum, which is not removed for a considerable time, amounting to months in some cases, readily retains food particles. Teeth only partly erupted, so commonly seen in mouth-breathers, are often affected very early in life. In these patients food may remain on the front surface of the incisors when the lip is retracted, so that the enamel over the whole surface may be attacked. The earliest change in the enamel is a loss of translucency, a chalky appearance replacing the bright tissue. It is necessary to distinguish maldevelopment (hypoplasia), in which there are pits and lines of deficient formation, usually on several teeth. As a rule, the enamel becomes perforated at one spot, which frequently, however, escapes detection when it is at the point of contact of two teeth. When the inner surface of the enamel is attacked, the destruction invades the dentine in two directions, along the tubules, and at its junction with the enamel. The rate of progress in one direction or the other is determined largely by the structure of the tooth. The poorer the development ("soft teeth") the more does the process spread beneath the enamel, while well-developed teeth ("hard teeth") show changes extending directly along the dentinal tubules, involving a small area and perhaps extending to the pulp with extraordinarily little destruction of the tooth.

The dentine is more rapidly destroyed than the enamel, and if the latter continues intact, a very large part of the crown may be involved before the disease is recognized; this is often expressed by the patient saying that the tooth was perfectly good, but suddenly broke to pieces. In these circumstances the affected part may be recognized by a white, chalky appearance seen through the still hard and polished enamel. Should the process be slower, the affected tissue becomes stained and the tooth has a dark brown or even black ap-

pearance; usually the enamel will have broken down when the staining is very pronounced.

The dentine is destroyed by a digestive process, the organisms entering the dentinal tubules, enlarging them until neighbouring ones are involved, and causing areas of destruction (liquefaction foci) which join one another. The whole carious patch is wedge-shaped, determined by the arrangement of the dentinal tubules, which radiate from the pulp surface to that under the enamel. The affected tissue is so soft in parts as to be capable of being washed away, while in others it is of the consistency of cheese; at times, when the process is very slow, or when there is a tendency to its arrest, the tissue may be leathery.

There is a lessened resistance to the force applied in mastication, and the friable tooth-substance breaks, leaving a cavity varying in size with the amount of destruction which has taken place.

The pulp is the next structure affected, an event which is of the utmost importance, for severe pain (*see* DENTAL PAIN) usually results, and sequelæ of great significance may supervene. Extension of infection from the pulp may lead to acute dental periostitis or periodontitis, i.e. inflammation of the periodontal membrane and the bone in the region of the apex. Food forced into the cavity is usually the determining factor, probably by preventing drainage rather than by actually forcing organisms through the apex.

The tooth is tender, particularly when bitten upon, or it is actually raised in its socket as the result of swelling of the periodontal membrane; momentary relief may be given during the early stages by clenching the teeth together. If drainage is not established, an acute abscess results, with all the symptoms and general disturbance of abscess in bone, and if unrelieved, nearly always perforates the outer alveolar plate and extends under the muco-periosteum (gum-boil) until the membrane is perforated and the pus discharged. If the tooth is allowed to remain untreated, a sinus results, which persists indefinitely, drainage occurring into the mouth.

When an abscess has once formed, a subsequent one may cause much swelling, with the characteristic oedema of the face but with little or no pain; the bone has previously undergone rarefaction, and consequently the osteitis is limited and less severe.

DENTAL CYSTS

The **treatment** of dental caries, apart from the relief of pain, can be carried out satisfactorily only by a dentist. When the pulp is exposed, as a temporary measure a piece of cotton-wool, just moistened with a solution made with resin 4 parts, carbolic acid 4 parts, and chloroform 3 parts, will be found valuable. Carbolic acid, creosote, oil of cloves, and many other substances are useful. When applying any of these, the cotton-wool should be introduced into the cavity with care, so that no pressure is applied to the pulp, otherwise acute pain will follow. With any indication of periostitis, recognized readily by pain following pressure or a slight blow upon the tooth, the cavity must not be plugged, as this prevents drainage, but the pulp chamber opened up as freely as possible. Tincture of iodine applied to the gum is helpful; when the pain is severe the liniment may be used. When suppuration occurs, drainage must be established. Extraction of the tooth effects drainage far better than any other procedure, but the dentist may use the pulp canals for this purpose; an incision through the gum may be necessary.

WARWICK JAMES.

DENTAL, DENTIGEROUS, AND MULTILOCLAR CYSTS.—Cysts associated with teeth are of these three types. All give rise to a swelling within the bone, causing absorption with formation of new bone over them. Each has a globular outline, which is irregular in the multilocular form. As they increase in size the bone becomes thinned, giving rise to characteristic "egg-shell crackling"; later, the bone is lost in part, and fluctuation is obtained. All are lined by epithelium, and are produced as the result of irritation. Some cells associated with tooth development have persisted; they proliferate, undergo degeneration, and produce a cyst.

Dental cysts are associated with carious teeth, and therefore occur usually in adults. *Dentigerous* cysts (i.e. a cyst with a tooth embedded in it) occur after the period of eruption. *Multilocular* cysts are less common, are more often found in adults, but may occur at any age. These various cysts do not give rise to discomfort unless they are large or become infected. They should be removed, as they cause deformity when large, and may displace neighbouring teeth. The removal of the epithelial lining is the essential factor in treatment to avoid recurrence.

WARWICK JAMES.

DENTAL PAIN

DENTAL PAIN (Dental Neuralgia).—Pain of dental origin is most commonly due to inflammation of the pulp, which becomes exposed as the result of dental caries. The pain is of an acute, darting character, worse when the patient lies down, and so disturbs sleep. It is usually located in the neighbourhood of the tooth, but frequently radiates over the face when it corresponds to the distribution of the second and third divisions of the fifth cranial nerve. From mandibular teeth the pain is referred to the branches of the third division; a molar frequently causes pain in front of the ear; pain from a third molar may radiate very widely, even to the shoulder. Pain from maxillary teeth follows the distribution of the second division of the fifth nerve, and radiates perhaps less widely than that from the mandibular teeth. Pain may be referred from the lower to the upper jaw, and vice versa, but never crosses the middle line. Unilateral pain in the face always suggests a dental origin. A diagnosis of neuralgia without detecting a cause should never be made. An inflamed or irritated pulp reacts most readily to a low temperature, such as cold water or cold air; a small pellet of wool moistened in cold water and applied to the neck of each tooth in succession will indicate the tooth affected. When degeneration of the pulp has occurred, the cold reaction is given less readily, but pain is more easily elicited on the application of heat. Pain caused by heat is associated also with a marginal osteitis produced by food packed between two teeth, or by a piece of displaced filling, or other cause. In this group the pain is less severe and of shorter duration than that from a pulp.

An inflamed pulp will react to other stimuli, pressure from food packed into a carious cavity being one of the commonest. Sweet, salt, and acid substances all act as stimuli to a pulp, but may also cause pain of a milder and shorter kind in the earlier stages of caries, when the sensitive dentine is exposed. The neck of a tooth denuded by abrasion from the toothbrush reacts in much the same manner, but the pain is of short duration. A rather more severe form of the same condition is found in teeth of which the necks become exposed by the destruction of the tissues by "pyorrhea alveolaris" or the recession of gum and bone which follow extraction of neighbouring teeth. A clasp upon a denture is rarely kept clean on account of the curve of the metal, though this may be easily accomplished by the use of the

DENTAL PAIN

charred end of a wooden match; the fine film of food ferments and renders the exposed surface of the tooth very tender. Pain from an inflamed pulp may cease or ameliorate when the patient sits up.

Acute pain may be associated with the eruption of a third molar (*see* ORAL SEPSIS). The pain of teething is akin.

The severe form of neuralgia known as trigeminal neuralgia major is probably of dental origin. This type of pain is associated almost always with the second and third divisions of the fifth cranial nerve alone. When peripheral stimuli are cut off by injections of alcohol into the nerve trunk, complete relief is obtained.

Pain following dental treatment, if severe, may result from irritation of the pulp, or from apical periodontitis due to filling a tooth in which the pulp was infected. Extraction of a tooth may be followed by pain; this is frequent in early stages of apical periodontitis, and ascribable to alteration of tension. The bone may be injured by careless extraction, the alveolar process (usually its outer plate) being fractured, either with or without fracture of a tooth. Marked thickening of the root of the tooth due to prolonged irritation may render the operation difficult and attended by greater injury; even when the socket is not fractured the bone is injured by forcing the bulbous root through a smaller orifice. A fractured tooth may cause no pain, but a prolonged attempt to remove the fragment remaining may lead to considerable injury of the bone. Should a portion of a pulp remain in the fragment, it may cause great pain. The pain may be due to infection of the tooth socket; in this connexion a mandibular socket may become infected by the falling into it of a fragment of tooth or of filling.

Treatment.—The relief of pain resulting from pulp infection is effected by removing from the cavity the substance (such as food or a filling) which is preventing drainage or even producing direct pressure. The relief may not be immediate, but it is usually effective in a short time. Immediate relief is given if pure phenol on wool is applied loosely in the cavity. Many other substances will give relief, such as oil of cloves, sodium bicarbonate or carbonate, creosote, and nitric acid. These applications either have an analgesic action or cauterize the exposed pulp surface, phenol doing both. Apical periodontitis is relieved by establishing drainage, food or any substance blocking the pulp canal being removed

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and local counter-irritants being applied to the gum. The patient may aid drainage by sucking upon the tooth. If relief is not obtained, the part should be fomented by hot water held in the mouth; water in which two or three poppy-heads to the pint have been boiled may be used. As soon as an area of softening under the gum is detected an incision should be made.

Pain of a less severe character, such as that of marginal osteitis, is best treated by syringing with a weak antiseptic, the mechanical washing being of chief importance. Substances packed between the teeth should be removed, and the patient directed to use the opposite side of the mouth and to avoid further injury to the part. The necks of teeth exposed by attrition from the tooth-brush are rendered insensitised by applying nitrate of silver or pure formalin, the latter being the more useful as it does not stain the teeth, although the former is perhaps more effective. A similar sensitive spot may be produced on the biting surface of a tooth, the attrition of biting and grinding causing complete loss of enamel and exposing the sensitive dentine.

WARWICK JAMES.

DENTITION AND DENTAL IRREGULARITIES.—In the tables on the next page the dates of eruption of the teeth are given.

A rough and simple method of remembering the dates of eruption of the permanent teeth, omitting the third molars, is as follows:—6, 7, 8, 9, 10, 11, 12 years. Then 6 and 12 are for the first and second molars and, counting from the middle line, 7 is for the central incisors, and so on, the canine, an exception, being at 11 instead of 9.

The most important teeth are the first permanent molars. They are the first to appear, they help largely to determine the relationship between the jaws, they are the most firmly implanted, are placed in the position most favourable for mastication, and possess the largest masticatory surface. Appearing between the ages of 6 and 7, they are frequently mistaken for temporary teeth; they present fissures upon their upper surfaces in which food readily lodges, and in consequence they often become carious. In a great number of cases these teeth are lost at an early age, and the whole mouth suffers an injury which is disastrous except in the few instances in which the neighbouring teeth come into contact without being displaced from their correct vertical position. As soon as the first molars

DENTITION AND DENTAL IRREGULARITIES

appear, the biting fissured surface should be brushed, last thing at night particularly, with a small stiff brush, and only a short movement should be made so that the bristles may enter the fissures.

There is little doubt that to the eruption of temporary teeth many troubles have been attributed which are due to other causes. The gum over an erupting tooth may be tender and swollen, obviously causing pain; and injury and infection of the tissue may follow the biting of any object which the child can procure. But disturbances attributed to "teething" need more scientific investigation; that they exist would appear to be established, but that they constitute for the prac-

ment in order to prevent almost inevitable dental disease. The simplest test is to see whether the posterior border of the mandibular canine is in contact with the anterior border of the maxillary canine when the jaws are closed; if so, the occlusion is sufficiently correct. Even with this relationship the arches may be so reduced that neighbouring teeth overlap, an irregularity which needs treatment.

Causes of irregular teeth.—Irregularity of the teeth is due to several causes, of which mouth-breathing is by far the commonest. Heredity undoubtedly plays a part. Some importance has been attached to large teeth appearing in small jaws, and vice versa; the latter is not often seen. Habits such as thumb,

PERMANENT TEETH

	<i>Incisors</i>		<i>Canine</i>	<i>Premolars</i>		<i>Molars</i>		
	<i>Central</i>	<i>Lateral</i>		<i>First</i>	<i>Second</i>	<i>First</i>	<i>Second</i>	<i>Third</i>
Dates of eruption in years—								
Maxilla	7½	8½	11½	10	11	6½	12½	} 17-25
Mandible	6½	7½	10½	10½	11½	6	12	

TEMPORARY, DECIDUOUS, OR MILK TEETH

	<i>Incisors</i>		<i>Canine</i>	<i>Molars</i>	
	<i>Central</i>	<i>Lateral</i>		<i>First</i>	<i>Second</i>
Dates of eruption in months .	5-9	8-10	9-18	12-20	20-30

itioner an easy escape in diagnosis is certainly true. Any irregularity in the arrangement of the teeth, or imperfection of their development, favours disease. With completion of the permanent set, the jaws should be well developed, the crowns of the teeth should project into the mouth quite free of the gum, which should be attached firmly to the necks, a "tongue" passing between each of the teeth to occupy the space completely. The crowns of neighbouring teeth should approximate so closely that food particles will not be forced between them during mastication. The whole arrangement should be so compact that the friction of biting cleanses and polishes the teeth, whilst any particles remaining can be removed readily by the tongue. The manner in which the jaws meet together—occlusion—is important, malocclusion demanding treat-

finger, tongue, lip, toe, and dummy-teat sucking give rise to almost characteristic deformities of the temporary dentition, which are nearly always transmitted to the permanent series. Loss of temporary teeth by extraction for dental caries will generally cause an irregularity of the permanent series. The absence of a tooth permits its neighbours to approximate, and the whole arch becomes flattened on that side. Delayed falling out of the temporary teeth rarely leads to malposition, but occasionally a second temporary molar becomes locked between the first permanent molar and the first premolar. The retention of a temporary tooth generally indicates absence of its successor or malposition, the permanent tooth at times remaining deeply seated in the bone. The teeth most likely to be absent are the third molars, the maxillary lateral incisors, and the

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premolars. The canines are those which most frequently fail to erupt or are retained deeply in the bone; the temporary canine may persist as late in life as the fortieth year. More unusual conditions which may cause malposition are injury, growths, cleft palate, etc. Treatment of the teeth in these cases can only be carried out by the dentist, in whose hands the cases should be placed at as early an age as possible.

Mouth-breathing is the most prominent cause of dental disease, for all mouth-breathers in due course suffer from pyorrhœa alveolaris, while dental caries occurs earlier and is more

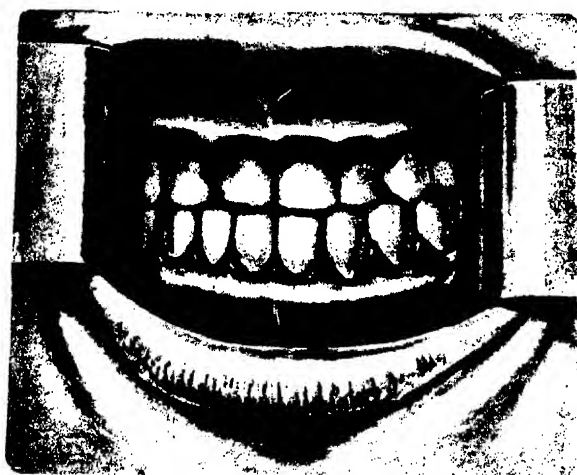


Fig. 19. — Showing lip line characteristic of mouth-breathing. The swollen gum margins correspond with the separation between the lips.

widespread; even the incisors become affected—a condition rarely seen otherwise in children. The position of the teeth and the relationship between the jaws is typical. Variations occur, but a description of the common form will suffice. The lips are apart, and, since they are not compressed, the upper curls upwards while the lower droops, the teeth and gums in consequence being exposed. The gum margin swells and, on examination, a definite line (Fig. 19) is seen where the healthier gum is marked off from the swollen part near the teeth. This feature is of great importance in diagnosis. It is met with also in a few patients who keep their lips apart but who are not true mouth-breathers. In order to allow air to pass through the mouth, the tongue is not in contact with the palate, and the two forces controlling the development of the maxillary

DERMATITIS EXFOLIATIVA

dental arch—viz. the tongue internally and the lips and cheeks externally—not being exercised, the high narrow palate results; and the teeth become irregular, for they, with the alveolar processes, are of fixed size, while the palate is under-developed, as is the whole base which supports it, including the nasal cavities with their surrounding bones. The incisor teeth usually project, approximating to the horizontal in some cases, a position mainly brought about by the action of the lower lip, which forms an elastic cushion on which they rest.

The mandible is altered in its relationship to the maxillæ; the mouth being open almost constantly, the weight of the tissues suspended from the mandible tends to displace it backwards, and to cause it to meet the upper jaw in a position known as post-normal. One of the worst effects is that the molar teeth meet sooner than they should, and in consequence have not sufficient space into which to erupt, so that they are not normally free of the gum.

Treatment of these cases must be directed to removal of the cause. Much can be done even in adults; the simple device of introducing a piece of rubber to prevent mouth-breathing at night is of great value. It is most comfortable when made to fit the mouth by a dentist.

WARWICK JAMES.

DERCUM'S DISEASE (see ADIPOSIS DOLOROSA).

DERMATITIS EXFOLIATIVA (*syn.* General Exfoliative Dermatitis, Pityriasis Rubra).—One of the more serious skin diseases, consisting of a more or less generalized inflammation of the skin associated with marked exfoliation. It may arise primarily or may supervene in some other chronic affection. It is comparatively rare, more common in males than in females, and may occur at any age in adult life.

Etiology.—The cause of the condition is unknown. It may appear quite suddenly in a previously healthy individual, or develop from a pre-existing psoriasis or scaly dermatitis. It is especially common in the individual who is subject to so-called chronic rheumatism, and joint-affections similar to those of rheumatoid arthritis are often associated with it. Local irritants, especially the too prolonged

DERMATITIS EXFOLIATIVA

use of chrysarobin in the treatment of psoriasis, may provoke an outbreak. A. G. Phear has described its occurrence as the result of taking quinine. Jadassohn found it frequently in association with tuberculosis, but the two diseases do not appear to have any direct connexion. Changes have been described in the nervous system, but whether they are the cause or the result of the disease is not known. No local parasite in the skin is known to be associated with the condition. Similarly alteration in the excretion of nitrogen and uric acid by the kidneys occurs, but owing to the great activity of the skin it is difficult to interpret these phenomena.

Pathology.—The microscopic changes in the skin throw practically no light on the condition, and vary with the stages of the disease. The chief change is hyperæmia with imperfect keratinization of the horny layer. In long-standing cases there is atrophy of the Malpighian layer of the skin with a disappearance of the papillæ. The glands of the skin may also be partly destroyed. From the histology there is no indication as to the origin of the condition.

Symptomatology.—Roughly, the cases of this disease may be divided into two classes, (1) the milder or seborrhœic type, (2) the severe type. In the first type it begins like an ordinary acute seborrhœic dermatitis affecting chiefly the flexures of the body and limbs, and rapidly spreads, involving large areas or even the whole skin. In the second type the whole body is affected, so that there is not a square inch of healthy skin anywhere. There are all varieties between the mild and severe cases, the varieties being rather a question of the degree of the affection than any difference in the disease. Once established, the condition tends to persist, always for weeks, often for months, and even for years. The whole skin becomes red, and the characteristic exfoliation is constantly going on. The erythema is of a peculiar yellow-red (salmon) colour, and not the pink-red colour seen in most skin eruptions. The surface is dry, and except for perspiration the skin is never moist and oozing. There is rapid formation of an imperfectly formed horny layer, resulting in the continual exfoliation of thin, greyish-yellow scales which are fairly easily rubbed off, leaving a dry red surface beneath. In severe cases handfuls of scales may be removed from the patient's bed daily. Sometimes there is considerable itching, but as a rule itching is not much com-

plained of. The chief complaint is usually a feeling of chilliness. This is due to the great evaporation which is taking place from the hyperæmic skin and the absence of a proper horny layer. Occasionally in the early stages there may be some rise of temperature at night, but later there is no rise unless some complication is present.

After the disease has lasted for some weeks or months the skin begins to get paler. This pallor usually starts in several areas simultaneously and the eruption very gradually fades. As the hyperæmia diminishes, the scaling also gradually disappears, till the skin returns to an apparently normal condition. It is not uncommon for exacerbations and remissions to occur. In many cases, after lasting for several months the disease disappears entirely, only to return some months or years later, so that the patient may have several prolonged attacks at intervals. It is exceptional for a patient to have one attack, which is recovered from, with no recurrence later. The worst cases are usually those associated with chronic joint-lesions of the nature of rheumatoid arthritis, and one such case I have seen which had lasted over forty years, without interfering very materially with the patient's general condition. But generally in the persistent cases sooner or later the general health suffers. From scratching, secondary infections, such as furunculosis, may occur. In severe and prolonged cases the nails may be shed and the hair fall out. As a rule the lymphatic glands are not enlarged, except where there is some secondary infection. There is no characteristic change in the blood. In chronic cases the process may invade the mucous membranes of the mouth, bronchi, and conjunctivæ, producing symptoms of irritation in these areas.

When the disease supervenes on psoriasis or scaly dermatitis, it is not unusual for the whole body to be affected with pityriasis rubra for some months, and when that disappears the psoriasis or scaly dermatitis returns.

Diagnosis.—This usually presents no difficulty. If the condition develops very suddenly, the diffuse erythema may suggest scarlet fever, but there are no throat symptoms, and the rapid appearance of exfoliation soon makes the diagnosis clear. *Erythema scarlatiniforme* and various drug rashes may occasionally look rather like pityriasis rubra on their first appearance, but a very short observation of the case will usually leave no doubt.

DERMATITIS EXFOLIATIVA

The disease which is most likely to be confused with it is *pemphigus foliaceus*. It is important to distinguish these two conditions, because in *pemphigus foliaceus* the prognosis is even worse than in *pityriasis rubra*. *Pemphigus foliaceus* and *pityriasis rubra* are the only two skin diseases which affect absolutely the whole skin. In the former there is a general redness with exfoliation of the skin, but it is quite different from what is seen in *pityriasis rubra*. It has not the yellow-red colour; it is pinker, and—what is more characteristic—if the scales and crusts are removed from the surface a moist oozing red area is left underneath. The presence of imperfectly formed blisters, and the ease with which the surface epithelium can be rubbed off, leaving a raw surface beneath, should serve to distinguish the two diseases. In *pemphigus foliaceus* also there is usually a very marked, sickly, or even stinking odour given off by the skin, whereas in *pityriasis rubra* there is absolutely no smell whatever. In *pemphigus foliaceus*, also, the *Bacillus pyocyaneus* can usually readily be grown in culture from the skin, and the blood shows a marked eosinophilia, neither of which is the case in general exfoliative dermatitis. An extensive *seborrhœic dermatitis* may also resemble *pityriasis rubra*, but in the former the whole skin is never affected, and there are always areas of vesiculation and oozing. *Seborrhœic dermatitis* is usually more itchy and the eruption not so uniform, being dry and scaly in one area, papular in another, and oozing in a third. The absence of any feeling of infiltration and thickening of the skin is a very marked feature of *pityriasis rubra*. In chronic scaly *seborrhœic dermatitis* the skin tends to become somewhat thickened, whereas in *pityriasis rubra*, the longer the condition lasts the more atrophic and thinned the skin becomes. Extensive *psoriasis* should also be easily distinguished by the character of the isolated spots and the fact that all the skin is not affected. Similarly widespread lichen planus is never universal, and isolated typical shiny papules are always to be found.

The **prognosis** should always be guarded, because, although the first attack may be recovered from, subsequent attacks will almost certainly occur. In a few cases the disease is associated with high temperature, and the patient dies in a few weeks in a cachectic condition, but as a rule it is the complications which are to be feared. Owing to the absence of a proper horny layer, which normally prevents

too rapid evaporation from the skin, these patients are very liable to chills, and pneumonia is especially common. If pneumonia does occur the result is nearly always fatal.

The **treatment** is not very satisfactory. The disease seems to run its course, whatever treatment is adopted. Internally there is no specific, but quinine, salicin, and sodium salicylate should be tried. Arsenic has not the effect which it has on pemphigus eruptions. Pilocarpin has numerous supporters, but its effects must be carefully watched. Antimony, in the form of vinum antimoniale 4 min., is also worth a trial. General tonic treatment, if indicated, should also be given. No form of dieting has any influence on the condition. The patient should be kept in bed. If allowed to go about he is very apt to catch cold, and as a rule the patients say that they feel most comfortable when in bed. With regard to local treatment, it should always be borne in mind that no strong or irritating application must be used. The tendency sometimes is, when the disease has remained more or less stationary for months, to use strong applications such as tar, etc., in the hope of clearing off the eruption. They always do harm, and may indefinitely prolong the attack. Only the very mildest applications are to be used. Lotions are not suitable. There is sufficient evaporation and cooling of the surface already, and whatever is applied should be of a greasy nature; the grease coats the surface and acts as an artificial horny layer. The patient should have a daily warm bath, and use a little plain soap to remove the accumulated scales and grease. If there is much irritation, a starch or bran bath should be given. After the bath a bland ointment should be rubbed all over the skin, and applied spread on strips of cloth bandaged on to the limbs. Considering the large area to be covered and the prolonged duration of the treatment, the question of the cost of the local application requires consideration. Plain vaselin, or olive oil, is often quite sufficient. An ointment consisting of paraffinum durum 2 parts and paraffinum molle 5 parts is also useful. If there is much itching, a paste such as the following is better than a simple ointment:—

R	Bism. oxychlorid.	gr. v.
	Paraff. moll.	ʒiv.
	Pulv. zinc. oxid.	} aa ʒii.
	Pulv. amyl.	

R. CRANSTON LOW.

DERMATITIS HERPETIFORMIS

DERMATITIS EXFOLIATIVA NEONATORUM (see IMPETIGO CONTAGIOSA).

DERMATITIS FROM EXTERNAL IRRITANTS (see ECZEMA).

DERMATITIS HERPETIFORMIS.—A disease characterized by an eruption of groups of vesicles in successive crops over periods of months or years, accompanied by intense itching. It has probably a close relationship with pemphigus vulgaris, with which it was formerly confused.

Etiology.—It may occur in both sexes at all ages, but it is most frequent in adult males. It was at one time thought to be of nervous origin, and cases following worry or shock were instanced in support of this view. The modern tendency is to regard the disease as due to some kind of toxin, perhaps a product of disturbed metabolism. A closely similar eruption, herpes gestationes, which may occur in the course of pregnancy, has been attributed to intoxication by the products of disassimilation of the foetus and placenta.

Pathology.—All that is known of the pathology of the disease is that, microscopically, the eruption is the result of an acute inflammation and that an excess of eosinophilic cells is found in the blood. No visceral lesions or nerve-tissue changes have been discovered.

Symptomatology.—The eruption is multi-form. The most frequent type is that in which there occur vesicles of the size of a pin's head to a pea, grouped in herpes-like clusters of two or more, or in rings or segments of circles. Instead of vesicles there may be small red papules, capped with fluid or ex-coriated by scratching. The vesicles or papules may be, but are not always, seated on an erythematous patch. In other cases the lesions consist of patches of erythema or of urticaria. Or, again, some of these erythematous patches may be the seat not merely of vesicles but even of bullæ, or pustules. In some cases the eruption may keep to one type, particularly to the most characteristic form of small grouped vesicles; in others the type may vary with different attacks; in yet others there may occur simultaneously erythema, grouped vesicles and bullæ. The various groups may extend peripherally and coalesce to form large irregular areas.

At first these eruptions may be scanty and in certain regions only, but soon or later they become widely distributed and sometimes

generalized. The mucous membranes may be involved, and the disease sometimes first shows itself in the mouth. The nail-beds are often affected, causing dystrophy or separation of the nails.

The eruption is not the most trying feature. Intense itching and burning sensations accompany it and make life a burden for the patient; and the scratching leads to excoriations and, perhaps, to secondary infections. These attacks may continue, with successive crops of eruption, for weeks or months, followed by an interval of complete or partial freedom for months or years, to be succeeded again by fresh attacks.

In spite of the severity of the symptoms and consequent want of rest and sleep, the general health is usually maintained, though malaise and fever are sometimes present during a severe attack.

Diagnosis.—A firm diagnosis may be very difficult until the patient has been observed through more than one attack—for recurrence is an essential feature of the disease. *Scabies* must, of course, be excluded, and careful search should be made for burrows and acari about the fingers and wrists. *Chronic urticaria* is distinguished by the fact that the eruption consists only of fugitive wheals, without any scratched papules or vesicles in groups. Chronic widespread *eczema* may bear some resemblance to dermatitis herpetiformis, but in place of superficial vesicles or bullæ, the patches of eczema show a thickening of the skin from congestion and oedema with, at some period, weeping and oozing. *Erythema multiforme*, at one time confused with dermatitis herpetiformis, is recognized by the symmetrical distribution of the eruption on the extremities and by the absence of itching. It may often be difficult to distinguish an attack of bullous dermatitis herpetiformis from the closely allied affection *pemphigus vulgaris*, and the subsequent course of the disease must decide the point.

Prognosis.—In most cases the attacks become gradually less severe and occur at wider intervals until they eventually cease. In some the eruption is almost continuous over many months or years, the patient is gradually worn out, and the disease ends fatally.

Treatment.—During a severe attack the patient should be confined to bed: a daily bran bath or bath of normal saline solution should be taken, and any raw surfaces dressed with a simple ointment. In cases which do

DERMATITIS, X-RAY

not yield to these simple remedies various drugs may be tried. In some cases arsenic acts almost as a specific, and some sufferers are able to cure or ward off an attack by taking this drug. Many cases are not helped at all by arsenic, and for these salicin or salicylates in fairly large doses may be employed, or quinine in large doses. The pruritus is sometimes relieved by phenacetin or by antipyrin. Locally, antipruritic lotions, such as lotio calaminæ, lead lotion, or a lotion of liquor picis carbonis, 1 dr. to 10 oz. of water, are often useful. Sulphur ointment vigorously rubbed in has given relief in some cases.

When these measures fail, one is justified in using blood-serum injections, which have lately been reported to have cured several cases. It is simpler, however, to reinject the whole blood than to centrifugalize and inject only the serum. Twenty c.c. of blood are withdrawn from the vein of a healthy person and injected intravenously into the patient, a proceeding which is repeated twice weekly. A still more simple technique, which has lately been employed, is to withdraw 20 c.c. of blood from the patient's vein and reinject into the muscles of the buttock. This blood-serum injection treatment is still on its trial, and should be reserved for obstinate cases.

H. G. ADAMSON.

DERMATITIS, SEBORRHOIC (see SEBORRHOIC DERMATITIS).

DERMATITIS VENENATA (see DRUG ERUPTIONS; ECZEMA).

DERMATITIS, X-RAY. — This affection may conveniently be divided into two clinical types, acute and chronic. The former results from a too intense exposure or aggregate of exposures such as a prolonged screen examination of a patient when the dose is not measured; the latter follows constant small exposures and affects operators who have not been adequately protected or patients whose malady requires repeated treatment extending over a period of many months or years. The one is an acute localized dermatitis comparable in many respects to an ordinary burn and its various degrees of intensity; the other, a slow insidious degenerative process tending to terminate in malignant changes. There is, however, an important difference between a burn due to heat and one due to X-rays in that the symptoms of the latter are preceded by a latent period varying from one to three weeks, or even longer,

according to the intensity of the dose and the condition of the tube; there are some cases on record in which several months or even years have elapsed before the development of the symptoms. An X-ray burn is also liable to become chronic, and is subject to periodical exacerbations.

Acute X-ray dermatitis.—About a fortnight after a normal maximum dose, as measured by tint B of Sabouraud's pastille, temporary epilation will occur without erythema and with only a slight, transitory reddening of the skin. A rather larger dose will cause permanent epilation, generally accompanied by a severe erythema and followed by an atrophic, pigmented, telangiectatic scar. A further stage is represented by the formation of vesicles or bullæ leaving a disfiguring telangiectatic scar. When the normal maximum dose has been greatly exceeded there will be ulceration involving all the layers of the skin and subcutaneous tissue, the resulting scar being depressed, shiny and vascular. Finally, total destruction of the tissues with sloughing and gangrene may occur. The duration of the reaction varies directly, while the latent period varies inversely, with the intensity of the dose administered. An X-ray ulcer is sharply margined, and sometimes limited by the clothing, such as the coat-sleeve; it is covered with an adherent yellowish membranous slough, and is acutely painful, and extremely slow to heal, resisting all palliative treatment. Further, it may become malignant.

Chronic X-ray dermatitis occurs almost exclusively in radiographers or their assistants or in tube-makers, and generally develops on the back of the hands, as these are the parts most exposed to the rays. It is due to the cumulative effect of small repeated doses, and is, of course, more likely to occur when adequate protective measures, such as the wearing of proper gloves, are not observed. In all probability there have been acute or sub-acute attacks of dermatitis as well, and these doubtless play no small part as a provocative factor. The exposed skin becomes dry, pigmented, atrophic, and covered with telangiectases, and the nails become reedy and brittle. By degrees hyperkeratosis ensues, localized warty thickenings forming on the affected skin, or there may be painful chronic ulcers with irregular margins. The nails and fingers are often livid and swollen, and very sensitive to cold. Finally, the warty excrescences or the ulcerated patches may slowly take on malignant characters.

DERMOID CYSTS

DIABETES INSIPIDUS

Treatment.—In acute cases soothing applications, such as linimentum calcis, should be prescribed, and if sloughs are present boric fomentations may be applied, if the heat can be borne. If the burn becomes chronic, excision and grafting or amputation may be required. In chronic dermatitis of operators, soothing ointments and pastes will help to protect the part and relieve pain. Carbon-dioxide snow has been used with success for the removal of warty growths; radium and high-frequency currents have also been tried. Excision and grafting or amputation are the only effective measures in progressive cases, especially when epitheliomatous changes ensue. Every precaution should be taken by operators to ensure proper protection and to avoid unnecessary exposure to the rays.

S. E. DORE.

DERMATO-MYOSITIS (see MYOSITIS).

DERMOGRAPHISM (see URTICARIA).

DERMOID CYSTS develop in those situations where two portions of the embryo normally come together and fuse. If the epithelium covering the opposing surfaces fails to disappear completely it may give rise to a dermoid cyst. Cysts also grow from the remains of some foetal duct which in ordinary circumstances does not persist. (For ovarian dermoids, really teratomata, see OVARIAN CYSTS.) Dermoid cysts are single, spherical, movable under the skin, and vary in size from a pea to a walnut; they appear during the first few years of life. They have a fibrous wall lined by stratified squamous epithelium. Hair-follicles with growing hair, sebaceous and sometimes sweat-glands are present in the wall. The contents consist of sebaceous material and hairs. Sometimes the contained material is watery or serous, and it may be chocolate-coloured from hæmorrhage. Growth is very slow. There are certain situations where dermoids commonly occur. In the *face* they are found at the outer angle of the orbit, root of the nose, or within the orbit. In all other possible positions between foetal facial processes they are very rare. In the orbit exophthalmos and interference with ocular movements are produced. The cysts situated on the margin of the orbit frequently lie in a depression of the bone, and may even be connected with the dura mater. In the *scalp* they are seen over the anterior fontanelle and external occipital protuberance, and in

these situations also are apt to have an attachment to the dura mater. About the *external ear*, just in front of the tragus and over the mastoid process are common sites, as is also the *floor of the mouth* just behind the incisor teeth (sublingual dermoid). In the last position a dermoid is distinguished from a ranula by the facts that it is strictly median in position and is yellowish in colour. It may also project under the chin, which a ranula never does. In the *neck* dermoids are found most often behind the angle of the jaw, where they are distinguished from glands by the fact that there is a single swelling, whereas a cold abscess in a gland has usually one or more smaller glands close to it. More rarely a dermoid occurs in the middle line of the neck in front. In the *trunk* dermoids are confined to the middle line of the body, and occur over the sternum, at the umbilicus, or in the sacrococcygeal region.

Treatment.—The cyst should be completely enucleated. If a portion of the wall be left, recurrence will take place. It must not be forgotten in operating upon scalp dermoids that they may be adherent to the dura mater. The removal of neck dermoids may necessitate a very difficult and deep dissection between the carotid vessels.

C. A. PANNETT.

DERMOID CYSTS OF OVARY (see OVARIAN CYSTS).

DEXTROCARDIA (see HEART, CONGENITAL DISEASE OF).

DHOBIE'S ITCH (see RINGWORM).

DIABETES, BRONZED (see HÆMOCHROMATOSIS).

DIABETES INSIPIDUS. — A chronic affection characterized by the passage of large quantities of normal urine of low specific gravity.

Etiology and pathology.—Only 15 cases occurred during ten years among more than 25,000 admissions to the medical wards of St. Bartholomew's Hospital. The disease is commonest in children and young adults, and is twice as frequent in males as in females.

It is clear that a number of different conditions have been described under this name, which is really merely that of a symptom. Persistent polyuria without the presence of albumin or sugar may result from (1) *syphilitic meningitis*, especially if it affects the base of the brain, (2) diseases of the pituitary body,

DIABETES INSIPIDUS

(3) a defect of the kidney rendering it incapable of secreting concentrated urine, (4) polydipsia.

(1) A strongly positive Wassermann reaction may be found in the subjects of diabetes insipidus who present no other evidence of syphilitic taint. Post-mortem examination has also shown syphilitic meningitis at the base of the brain.

(2) The cases of pituitary origin are the worst, because they usually depend on a new growth, though caseous tubercle in this situation may be responsible. The internal secretion of the posterior lobe of the pituitary body is disturbed. It is quite probable that slighter conditions, such as the temporary polyuria following head injuries, may depend on transitory lesions of the pituitary gland. As over-secretion of the gland can be produced by nervous stimuli, hysterical polyuria may be due to a similar cause. It is probable that basal meningitis may produce its effect through irritation of the gland.

(3) In the form associated with the primary defect in the kidney, this organ is incapable of secreting urine of the usual concentration, so that a much larger quantity of water has to be excreted to remove normal metabolic products. Consequently 20 grm. (5 drachms) of sodium chloride will cause a greatly increased diuresis in this, but not in the other types, in which the concentration of the urine can temporarily be increased; also the whole of the administered salt will not be excreted within twenty-four hours (Meyer). In interstitial nephritis also there is a diminished capacity for secreting concentrated urine and salts, and in some post-mortem records of diabetes insipidus the condition of the kidney certainly resembles that of chronic interstitial nephritis. It would not be easy to recognize a minute trace of albumin in the large quantity of water excreted. Meyer has elaborated another test for this variety of diabetes insipidus. Theocine-sodium-acetate increases the permeability of the kidney and thus acts as a diuretic in the normal individual. In this type of the disease, however, an increased permeability of the kidney renders the excretion of so much water unnecessary, so that the drug may actually diminish the polyuria for a time. The blood-pressure is likely to be raised as in other lesions of the kidney. Even this type the pituitary body may be responsible. Morley Fletcher has described a case of infantilism with interstitial nephritis-recrudescence rickets; the pituitary

body may be enlarged. This would approximate to the renal type of diabetes insipidus. That cases described as diabetes insipidus sometimes depend on a kidney lesion is further shown by Saundby's observation that the cause of death may be the gradual destruction of kidney tissue producing uræmia. But it appears to me that they should not be included under the present heading.

(4) In the cases showing none of the above associated symptoms it has been thought that the polyuria is secondary to the acquired habit of drinking large quantities of fluid. But the restriction of the fluid intake to a normal level should produce a normal output, even though causing great thirst. Instead of this, although the level of excretion falls, it does not drop to normal, and the tissues become desiccated. A more reasonable view appears to be that just as the pituitary body may be irritated by meningitis or new growth, so it may be stimulated by its sympathetic nerve supply, as described by Cushing and others. The influence of shock and emotion in producing the disease is of interest in this connexion.

Symptomatology.—The patient may wake up one morning feeling very thirsty, polyuria from that time being established. Injury or fright may cause an equally sudden beginning. More commonly the polyuria comes on gradually. The urine is increased more than in diabetes mellitus, eight pints being usually passed in a day, whilst sixteen to twenty-five pints are not rare. It is very pale in colour, faintly acid or neutral, and soon becomes turbid and alkaline. The specific gravity may be only 1002 or 1003, but even this may represent an increased excretion of solids, allowing for the greatly increased total quantity of fluid. Occasionally calcium oxalate and phosphate crystals and muscle sugar (inosite) may be present. If a trace of dextrose appears as the case develops, it is certainly of pituitary origin. The skin is dry and rough and the quantity of sweat diminished. The thirst is intense: I have known a patient drink six syphons of soda-water at one time. The subjects of this disease will sometimes, if deprived of water, drink the contents of the washing basin or even their own urine. Digestion is usually satisfactory until the general health fails, when there is anorexia. The tongue becomes red and glazed; the throat and mouth are coated with sticky mucus and the breath becomes fetid. The bowels are usually con-

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stipated, but there may be diarrhoea in the final cachexia; the temperature is generally subnormal; the pulse is small and easily compressible, except in the renal type, in which the artery becomes hard and the blood-pressure may rise as high as 150 in a child. Various nervous symptoms are common—lumbago, sciatica, muscular cramps, pruritus, hiccough, headache, impotence, and insomnia. Ocular signs and symptoms, lethargy and drowsiness, occur in the pituitary type.

Diagnosis.—The condition is easily distinguished from *diabetes mellitus* by the absence of sugar and the low specific gravity. *Hysterical polyuria* is intermittent in its occurrence, and accompanied by other hysterical manifestations. Actual measurement of the amount passed is necessary to distinguish the condition from one of mere *frequency of micturition*. *Fraud* can be detected by keeping watch on the amount of fluid drunk, as it is impossible for anyone to continue to excrete such large quantities without drinking copiously. The most important point is the differential diagnosis between the different conditions causing the symptoms of diabetes insipidus. A Wassermann reaction is essential. Cases with ocular disturbances such as bitemporal hemianopsia and optic atrophy, and later drowsiness and coma, are almost certainly of pituitary origin. The head should always be skiagraphed, the rays being exactly transverse to the clinoid processes. Enlargement of the pituitary fossa would settle the question. The reaction to sodium chloride and to theocine-sodium-acetate should also be determined. In this way the case may be allocated to one of the first three groups described. If it belongs to the fourth category, i.e. primary polydipsia, the effect of cautious restriction of fluid will soon establish the diagnosis.

Prognosis.—In the light of fresh knowledge of the various conditions causing diabetes insipidus, our views as to the prognosis must be recast. The sinister view generally held as to the outlook is probably based on examples of the pituitary form, which certainly tend to get worse rapidly because these patients are generally suffering from a new growth. The cases of primary kidney incapacity go downhill, though more slowly, because of the progressive destruction of kidney substance. On the other hand, persistent polyuria dependent on specific meningitis or its results may last for many years, and yet in other respects the patient may enjoy good health. Should,

however, the meningitis spread, the prognosis becomes that of ordinary cerebral syphilis. Polyuria of sudden onset or following head injuries may subside quite rapidly. It remains true, however, that a guarded prognosis has to be given in all cases until the response to treatment has been determined. Death may occur from coma in the pituitary type, from uræmia in the renal type, and from exhaustion or intermittent affections in any type.

Treatment.—In the first group, vigorous antisypilitic treatment should of course be employed. A drachm of mercurial ointment should be rubbed in daily and rather full doses of potassium iodide given, starting with 5 gr. three times a day and increasing to 15 gr. or even more. Sometimes the best effect is obtained by giving full doses of iodide for a fortnight at a time, with intervals of a fortnight's abstinence. In the second group, little can be done beyond symptomatic treatment. As in many conditions of new growth, temporary alleviation can be procured sometimes by iodide of potassium. If the growth appears to be getting larger, as evidenced by headache, vomiting, drowsiness, and increasing ocular signs, operation may be considered, but at best it is a desperate expedient. In the third group, measures should be taken to diminish the excretion of solids by the kidney. The amount of salt should be rigidly restricted and all meat extracts forbidden. The amount of protein should be reduced. The patient can be encouraged to satisfy his needs with carbohydrates and fats, which will diminish his appetite for nitrogenous food. Meat should not be allowed more than once a day, and then only in small amounts. Tea, coffee, and alcohol should be prohibited. The question of restriction of fluid intake is an important one. Clearly, in the third group, to restrict fluids is sheer cruelty, as the kidney is incapable of excreting a concentrated urine. In such cases, the restriction is followed by loss of weight and a rise in the output of nitrogen as the deprivation of water causes a breakdown of the tissues. This increased excretion of nitrogen in turn demands more excretion of water. The tissues are drained, and the thirst becomes intolerable. The appetite and the general health will soon seriously deteriorate. In the first and second groups restriction of fluid, whilst not so dangerous, is usually ineffective. In such cases, the patient should be told not to gratify his thirst needlessly.

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but should not be definitely restricted except on the following plan. If the fluid ingested and excreted is approximately the same, the amount of fluid drunk should be reduced by one to two pints. If this is followed by a diminution in the urine, the fluid can be further reduced every third day, but this procedure must not be continued after the urine has ceased to diminish. Ralfe suggested this plan for the treatment of diabetes insipidus in general, but, from what has already been said, it is clear that it is only likely to be effective in the fourth group, i.e. when polyuria is secondary to polydipsia.

At best the treatment must be largely symptomatic. Acidulated drinks, or half an ounce of raw oatmeal stirred into two pints of water and flavoured with lemon, are pleasant. Thirst may be temporarily alleviated by frequently rinsing the mouth with water without swallowing it. Whatever the origin of the case, tea, coffee, alcohol and salts are inadvisable.

Valerian is the drug which has the greatest reputation in the symptomatic treatment, and I have found it a great help even in the syphilitic cases. As much as half an ounce of the tincture has been given four times a day, apparently with success, but few patients are likely to tolerate such heroic doses. In smaller amounts it may be reinforced by the addition of some bromide of ammonium and 5-10 min. of tincture of cannabis indica. Valerianate of zinc has also been used in large doses (15 gr., gradually increasing to 30, three times a day). With smaller quantities (9 gr. in a day) I have sometimes noted that while the output falls the intake rises, which suggests that the drug acts largely by retaining water in the tissues; consequently it can only be effective in such cases for a short time. The effect of ergot (10 min. of the fluid extract or tincture thrice daily) is usually transitory, and a careful watch must be kept for symptoms of ergotism. Arsenic and iron may help as tonics. Electrical treatment with the constant current, between the nape of the neck and the naso-pharynx, has been recommended, but I have no personal experience of its doing good. In the pituitary type lumbar puncture has afforded relief. It is clear that, as with prognosis, our ideas of treatment must undergo considerable modification with the recognition of the fact that diabetes insipidus is probably produced by various causes acting on the pituitary body. An injection of 1 c.c.

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of pituitrin will check polyuria for several hours, but it can hardly be given so repeatedly as to form an effective treatment.

W. LANGDON BROWN.

DIABETES MELLITUS.—A disturbance of carbohydrate metabolism resulting in glycosuria, accompanied in the severer cases by thirst, polyuria, wasting, and imperfect oxidation of fats.

Etiology.—During a period of ten years, 253 cases were admitted to the medical wards of St. Bartholomew's Hospital, of whom 182 were males. There were no cases of patients under 5, and very few of patients under 10. The maximum incidence was between the ages of 20 and 30, but this is partly due to the fact that the disease assumes a milder form in later life, so that more are treated as out-patients. Hereditary influences appear to be important. Jews are specially liable to the disease, and families with a tendency to glycosuria not infrequently possess a Jewish strain. Apart from the Jews, subjects of diabetes are usually of a fair or sandy complexion. The disease is rare in negroes. It is commoner in the well-to-do than in the hospital class. Mental shock, nervous strain, and worry often seem to determine the onset. Gout is regarded as a predisposing cause, but probably merely because elderly fat persons are liable to both gout and alimentary glycosuria. The obesity which sometimes precedes glycosuria is probably a sign of deranged metabolism, which is compensated at first by storing the excess of sugar in the blood as fat. Syphilis may be responsible, by producing a pancreatic lesion. Injuries to the brain or spinal cord appear to start the disease sometimes, but a definite intracranial lesion should be regarded as a cause of symptomatic glycosuria rather than of true diabetes. Alcoholic excess produces glycosuria, usually of an amenable type. "A combination of intense application to business, over-indulgence in food and drink, with a sedentary life, seems particularly liable to induce the disease" (Osler).

Pathology.—Systematic analyses of the sugar in blood, which recent methods have made easy, show that in almost all forms of glycosuria there is hyperglycæmia, the normal 0.1 per cent. rising to 0.3 or even 0.4 per cent. The exception to this is "renal glycosuria," in which, as in phloridzin poisoning, there is hypoglycæmia, the kidney being unduly permeable to sugar. In such cases

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there are no symptoms, the condition being usually detected in the course of a routine examination such as that for life insurance. The glycosuria is slight in amount but constant, and is not affected by dieting. Indeed, the general health, which otherwise remains good, is impaired by strict diet. In three recent cases of mine of this type, the percentage of blood sugar was found to be 0.04. Apart from this, hyperglycæmia is the rule, and it may outlast the glycosuria. My own impression is that it generally develops as the disease progresses, and is not an early symptom. Probably it is a defensive mechanism adopted by the body to prevent the escape of a valuable foodstuff. But its retention in the blood-serum has drawbacks, as indicated below.

The glands which control carbohydrate metabolism fall into two antagonistic groups; the first consists of the pancreas, whose internal secretion promotes the utilization of sugar by the tissues, and increases carbohydrate tolerance; the second comprises the thyroid, the pituitary, and the suprarenal, the secretion of each of which mobilizes the sugar into the blood and diminishes carbohydrate tolerance. The members of the second group have two other features in common besides this effect on carbohydrate metabolism; they are all associated with the activity of the reproductive organs, and they all have their secretion controlled by the sympathetic. It is the correlation between this group and the reproductive glands that accounts for the influence of pregnancy in exciting glycosuria.

The main rôle of carbohydrate in metabolism is to furnish fuel for muscular energy and to provide for the complete combustion of other foodstuffs, particularly the fats.

The pancreas comes into activity when food is being prepared for absorption into the body; its external secretion is therefore pre-eminently concerned in the storage of energy; the internal secretion acts in the same direction. The action of the pancreas is anabolic and, like other anabolic activities, is controlled by the vagus, though not to the same extent as some of them. And as the vagus and the sympathetic are opposite in effect when supplied to the same structure, we should expect that the sympathetic would be inhibitory to the pancreas as it is to other digestive processes. The antagonistic group, like other structures controlled by the sympathetic, come into action when preparation is being made for display of energy.

Sympathetic stimulation is in the primitive state a preliminary to fight or flight. "Emotion moves us, hence the name," says Sherrington. Perhaps it would be more correct to say that emotion should lead to movement. But under conditions of civilization the response to emotion tends to be repressed, while preparations for that response still occur. Among these preparations is the mobilization of blood sugar, which is required for the anticipated display of muscular energy, since active muscle consumes three and a half times as much sugar as resting muscle.

Nervous energy tends to run in accustomed channels. Hence the influence of training. But this applies equally when the nervous energy is perverted; the emotional stimulus may persist because the natural response does not occur and the increased blood sugar becomes habitual.

It is clear, then, that anything diminishing the secretion of the pancreas or increasing the secretion of its antagonists will lower sugar tolerance and may excite frank glycosuria. Now, any of the glands controlling carbohydrate metabolism may, of course, become the seat of organic disease. But in that case there will be other signs besides the effect on carbohydrate metabolism. If the pancreas be defective there will be fatty diarrhoea and probably muscle nuclei starch grains will be present in the stools. If its duct be obstructed, excess of its diastatic ferment will pass into the urine, where it can be identified. If its tissue cells are disintegrating, some of the products will also be found in the urine. The effects of hyperthyroidism are far-reaching and easily recognized. Over-activity of the pituitary may show itself in skeletal changes, polyuria, and pressure effects on the 2nd and 3rd cranial nerves. Clinically we know less of over-secretion of the adrenals in disease, but we might expect it to be accompanied by general sympathetic irritation and raised blood tension as well as by glycosuria.

It must be admitted that improved diagnostic methods of recognizing signs of disease in these glands have not led to their being found in an increasing proportion of cases of clinical diabetes, although glycosuria may accompany organic disease of these glands. This led to the promulgation of the polyglandular hypothesis to explain diabetes. It was regarded as due to a "loss of balance between internal secretions." But how is such a loss of balance brought about? One can understand a loss

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of balance in a tripod if one leg is broken off. When one gland is diseased the antagonists will show relatively increased activity, just as a group of muscles will show contracture when their antagonists are paralyzed. But when none of the glands is organically diseased, it seems to me that the only way in which a loss of balance can be produced is through a disturbed innervation. And it is clear that sympathetic irritation will at the same time diminish the activity of the gland which promotes sugar utilization and increase the activity of the group of glands which throw sugar into the blood.

No explanation of diabetes is adequate which leaves the sympathetic nervous system out of account.

Allen, although one of the most convinced supporters of the pancreatic origin of diabetes, admits that diabetes may proceed to a fatal issue and still leave the pancreas as good as normal. He is, therefore, obliged to postulate the existence of some nervous action, which is, as he says, more probably irritative than paralytic. Such an irritative action of the sympathetic would, as I have shown, supply the requirements of his postulate. As the effect of the sympathetic is less pronounced in diminishing the activity of the pancreas than in increasing the activity of its antagonists, it does not seem likely that its action would be confined to the pancreas, especially when we remember that the sympathetic nervous system is anatomically designed to produce widespread effects. Allen is opposed to the idea of an antagonism between the pancreas and the other group, although his own experiments appear to me to support that hypothesis.

No attempt has here been made to draw a hard-and-fast rule between glycosuria and diabetes, for, apart from renal glycosuria, it is doubtful if such a distinction can or ought to be made. A normal individual may pass sugar after ingestion of 150-200 grm. ($4\frac{1}{2}$ -6 $\frac{1}{2}$ oz.) of sugar at one time, but a man who "passes sugar after the free taking of starch is virtually a diabetic" (Naunyn). Every grade between alimentary glycosuria and severe diabetes exists, and a mild case may become severe. Unfortunately it is less common for a severe case to become milder.

The realization of the significance of acetonaemia has profoundly modified our conception of diabetes. If the body is deprived of carbohydrates, or is unable to utilize them, it has to live on its fats to a considerable extent.

In the absence of carbohydrates the fats are incompletely oxidized to diacetic acid, the source of the acetone in the urine and breath. According to Hurtle, the formation of β -oxybutyric acid is an attempt on the part of the body to combat the toxic effect of diacetic acid by converting it into a more saturated and less poisonous form. These abnormal acids stimulate a protective formation of ammonia by the tissue proteins, and are therefore excreted partly as ammonium salts, nitrogen being lost thereby. They also combine with metals, and thus drain the body of calcium and magnesium. The similarity between diabetic coma and the results of the experimental injection of acid is so close that it is not surprising that the symptoms of coma have been referred to acid intoxication, especially as abnormal acids are known to be present in the circulation, although the comparative failure of alkalis to relieve the coma has been admitted. Yet in coma the alkalinity of the blood need not be reduced, and the experiments of Hurtle and Trevan show that diacetic acid is toxic apart from its acidity. Thus sodium diacetate will produce the symptoms of coma. It is this intrinsic toxicity of the diacetic acid which stimulates the respiratory centre, producing the characteristic air-hunger, which leads to sufficient over-ventilation of the lungs to reduce the CO_2 in the alveolar air from the normal 5 per cent. to 3 per cent. or lower. The acidosis resulting from the formation of diacetic acid may thus even be over-compensated by (1) protective ammonia-formation, (2) increased excretion of acid, (3) increased ventilation of the lungs, and yet coma may supervene. Although the formation of diacetic acid occurs in a number of conditions having in common the non-assimilation of carbohydrates, the compensatory measures adopted by the body are usually adequate to prevent toxic symptoms except in diabetes. The large consumption of protein and fat formerly advocated in the treatment of diabetes is now recognized as a potent excitant of coma.

Morbid anatomy.—Necropsy sometimes reveals very little. The blood is generally unduly bright in colour, and may often show abundant globules of fat or lipoids, which rise to the surface, forming a creamy layer (lipæmia). This excess of fat may even produce fat-embolism in the pulmonary vessels. Gross lesions of the brain are evidence of a symptomatic glycosuria rather than of true

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diabetes, but there may be multiple peripheral neuritis or sclerosis in the posterior columns of the spinal cord. The heart is small unless there has been arterio-sclerosis also. If there is peripheral gangrene the vessels generally show arterio-sclerosis. There is no constant alteration in the liver, except in the rare condition of bronzed diabetes, when it is large and cirrhotic. The pancreas may show no change, even microscopically, but interstitial pancreatitis with hyalin degeneration of the cell islets is comparatively common. The stomach may be dilated from habitual drinking of much fluid, and the rectum is generally loaded with feces. The kidneys are often hypertrophied, and may be fatty.

Symptomatology.—Though, exceptionally, the onset of the disease appears to be attributable to shock, it is usually insidious. Considering how frequently the condition is first discovered by examination for life insurance or as the result of ocular trouble, we are justified in concluding that it usually exists for some time before causing symptoms. Probably the glycosuria is intermittent before it becomes persistent. Thirst, polyuria, and wasting are the leading symptoms. The urine is increased to about four pints in the mild cases, and may amount to eight or ten pints in the severe. It does not attain the extreme limits often found in diabetes insipidus, and glycosuria can exist without polyuria. The urine is clear and pale, sometimes with a slight greenish tinge. The specific gravity is usually between 1030 and 1045, but it must be emphasized that a low specific gravity does not exclude glycosuria. Failure to recognize this truth frequently leads to glycosuria being overlooked. There may be various reducing substances present in the urine, such as glyconic acid, uric acid, and kreatinin, besides other sugars than dextrose. If, however, equal quantities of boiling Fehling's solution and urine yield an orange precipitate on mixing, without reboiling, dextrose is almost certainly present. Confirmation may be obtained by boiling ten parts of the urine with one part of Nylander's reagent for two minutes, when a black precipitate indicates sugar. To establish beyond doubt that the sugar is dextrose it is occasionally necessary to apply the fermentation, polarimeter, or osazone tests. The amount may be estimated by Fehling's method or by fermentation (*see URINE, EXAMINATION OF*). These quantitative methods are sufficiently accurate for clinical purposes. Of

the improvements on Fehling's method, I regard Benedict's (*see URINE, EXAMINATION OF*) as the quickest and simplest, while Citron's is probably the most accurate.

The amount of sugar varies greatly; the excretion of 200 grm. (3,000 gr.) in a day would indicate a severe case, but larger quantities than this are not at all common. It is essential that the presence of diacetic acid should be looked for (*see URINE, EXAMINATION OF*). It is doubtful if acetone is present in more than traces in the urine. Wasting is present in the severe cases, and diacetic acid is certain to be found, since the patient is living on his fats. The prognosis is then more serious.

A raw, glazed tongue, denuded of its superficial epithelium, is typical of severe diabetes. Less commonly, especially in stout subjects, a black fur forms towards its base, the so-called "hairy" tongue being produced. Dental caries is very liable to occur, and the saliva may be acid instead of alkaline. The breath may smell of acetone, and a sweet heavy odour is not uncommon. The patient may complain of a sweet taste in the mouth. Usually the appetite and digestion are good, and the former may be voracious, enabling the patient to tolerate the monotonous diet. If severe symptoms of acid dyspepsia occur with glycosuria there is probably a definite pancreatic lesion. Owing to the loss of fluids, the skin becomes harsh and dry, the hair thin and the nails brittle. Often there is a flush on the cheeks which spreads to the roots of the hair. In spite of the diuresis, the heart does not usually hypertrophy nor does the blood-pressure rise. The cases in which there is hypertrophy of the heart with high blood-pressure, and albumin as well as sugar in the urine, fall into a separate category.

Muscular weakness is common, and fatigue is readily induced. Mentally there is usually depression, and the patient is sometimes irritable and suspicious.

Complications.—Important changes may occur in the **skin**. Carbuncles or crops of boils are common, and the former are dangerous. Indurated, rounded, or conical swellings of a dull-red colour with yellow tips, to which the name of xanthoma has been given, may appear. Gangrene is apt to follow slight injuries, particularly in older subjects, in whom also arterio-sclerotic changes are almost invariably found, the combination of a defective blood supply with saccharine tissues affording an excellent medium for bacteria.

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Perforating ulcers may form in the feet when neuritis is present. Little centres of necrosis may start in the sweat-glands about the ankles or dorsa of the feet, and, unlike perforating ulcers, may develop while the knee-jerks are still present. Pruritus vulvæ may be the first symptom to call attention to the glycosuria; it is set up by yeast and other organisms which flourish in the saccharine urine. It causes great discomfort, and may lead to eczema and troublesome dermatitis. Balanitis in men is less common.

The **nervous system** generally becomes affected in severe cases. Neuralgic pains in the arms and legs are very common, and there may be definite peripheral neuritis with loss of knee-jerks. Far less commonly the neuritis assumes a severe form and imitates tabes: lightning pains, ataxia, crises, loss of reflexes, and perforating ulcers all occurring. The pupil, however, is usually spared. Degeneration of the posterior columns may be found post-mortem in such cases. Occasionally a laryngeal palsy results from neuritis. The mental alteration described above sometimes deepens into insanity.

Of the *organs of special sense*, the eye is the most likely to suffer. Ophthalmoplegia may occur and is apparently due to neuritis of the third nerve. The prognosis is not serious. A soft cataract may form. Sudden amblyopia or a central scotoma may develop. This is nearly always in smokers, even though they may have been quite moderate, so that it is probably due to the combined action of diabetes and tobacco. When tobacco amblyopia occurs in a moderate smoker it is well to examine for glycosuria. Lipæmia may be obvious in the retinal vessels. Retinitis with hæmorrhages may be found; although this is more likely to occur when there is a kidney lesion as well, it may arise in simple diabetes.

Diabetic collapse.—Sometimes the patient suddenly has an alarming collapse. There is pronounced cyanosis, while the pulse becomes small and very frequent. The patient becomes drowsy and may die within a few hours, and yet there may be no evidence of acid intoxication. R. T. Williamson, who has called special attention to this dangerous complication, attributes it to myocardial degeneration. It is commonest in elderly subjects.

Respiratory complications are always dangerous in diabetes. Even if the crisis is successfully passed in pneumonia, gangrene of the lung may follow. Phthisis is a common com-

plication, and generally runs a rapid bronchopneumonic course; the sugar may disappear from the urine spontaneously as the tuberculosis advances.

Renal complications.—Albuminuria is frequently associated with glycosuria. If the glycosuria is accompanied by hypoglycæmia it has little significance. In "gouty" glycosuria there may be raised tension, cardiac hypertrophy and albuminuria. Alcoholic excess not infrequently leads to the combination of glycosuria and albuminuria. If the alcohol is restricted the glycosuria tends to clear up, though the albuminuria may prove more refractory. On the other hand, prolonged glycosuria is very apt to lead to albuminuria, due, according to Pavy, to irritation of the kidney. The appearance of casts without albumin in the urine of a diabetic patient is always serious, and is regarded as a warning of coma.

Glycosuria in pregnancy.—Diabetes generally causes amenorrhœa, and conception is rare. When it occurs the fœtus is likely to die, and hydramnios is often present. It is therefore unnecessary to consider the interests of the child as against those of the mother, and unsatisfactory symptoms warrant a termination of the pregnancy. It is essential to be sure that the diabetes preceded gestation, since glycosuria of a much more amenable type may be due to pregnancy itself, clearing up after delivery, and perhaps recurring with subsequent pregnancies. Though usually of favourable prognosis, it may lead to true diabetes.

Many of the symptoms of diabetes are produced by the hyperglycæmia. This causes a rise of osmotic pressure in the blood which drains the tissues of fluid; hence the thirst and polyuria. Hyperglycæmia is generally believed to cause the neuritis, and there is no doubt that the serious skin infections are due to the saccharine condition of the tissues. It is probably also a factor in inducing cataract. Diabetic coma, however, is the result not of hyperglycæmia but of the toxic action of diacetic acid.

Coma may terminate the case at any age, but it is commoner in the young. Careful attention should be paid to premonitory symptoms, as it is possible to do much to avert coma, whereas nothing really effective can be done when it has fully developed. Clinical pathology has provided us with two valuable indications, the diminution of alveolar CO₂ and the rise of ammonia nitrogen. The formalin method is a fairly simple way of

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estimating the latter. I have never known a patient sink into coma if the ammonia remained consistently low, i.e. under 1·5 grm. of ammonia nitrogen a day; but when, despite treatment, the ammonia increases to 3 grm., the patient is liable to coma at any time, and a rise to 4 grm. is an almost certain indication of its rapid onset. Sometimes there is a sudden fall in the excretion of sugar. To restrict a diabetic's diet suddenly to protein and fat is to court this disaster, especially if he is kept entirely in bed at the same time; on the other hand, immoderate muscular exercise may be responsible for the catastrophe. A "bilious attack" may be a premonitory symptom, whilst epigastric pain or a burning sensation in the throat is very suggestive. Obstinate constipation is common, and a carbuncle may excite it. The patient becomes drowsy, and the characteristic dyspnoea, or air-hunger, declares itself. He soon loses consciousness, his respiration being deep and sighing. The face is pale, not cyanotic, the extremities are cold, and the temperature is subnormal. The pulse is small and frequent. The odour of diacetic acid in the breath, which has been compared to that of apples packed in hay, is very marked. That of acetone is more acrid and disagreeable. Death generally occurs within forty-eight hours of the onset of coma.

Diagnosis.—The first point to be determined is whether a reducing substance found in the urine is really dextrose. Confusion is not likely to occur if the precautions described above are observed. Glycuronic acid may cause a reduction, but does not ferment. It is usually present as a result of the administration of some drug, such as opium, chloral or camphor. I have found it after codeia. It is easy to see how this may cause mistakes. Of other sugars than dextrose, the commonest is lactose, which is often present in the later months of pregnancy, during lactation or at its termination. It does not ferment within twenty-four hours, and yields an osazone with considerable difficulty. Lævulose seldom occurs by itself, though there is lowered tolerance of lævulose in diseases of the liver. Maltose is seldom found, whilst pentose, which is a sign of an inborn error of metabolism, is extremely rare. If the reducing substance is dextrose, we must exclude *temporary glycosuria* due to head injuries, administration of anæsthetics or excess of thyroid or suprarenal extract. The *symptomatic glycosuria* of cerebral tumours, acromegaly, exophthalmic goitre, and diseases of

the pancreas must also be excluded. After cerebral hæmorrhage the urine is apt to contain sugar, and if the patient is brought comatose to a hospital, the diagnosis of diabetic coma is very apt to be made. The mistake can readily be avoided by testing for diacetic acid, which will always be present in diabetic coma, but not in a recent cerebral hæmorrhage.

If the amount of sugar passed, though small, does not yield to dieting, "renal" glycosuria may be suspected, and the sugar in the blood should be estimated.

Prognosis.—In general terms, the younger the patient the more rapid the course, though von Noorden has described some cases of amenable glycosuria in children after measles, diphtheria, and tonsillitis. It is extraordinary how well a child with diabetes may seem until the onset of coma. The presence of acetonuria always increases the gravity of the prognosis, since it shows that the metabolism of proteins and fats is also disturbed; but it is not the case, as was formerly thought, that acetonuria in diabetes foretells death within two years. A high ammonia output is of more significance than a deep colour with the nitro-prusside test. From the age of 40 onwards the disease runs a milder course. After this age, if there is not wasting or acetonuria, if the knee-jerks are present, if moderate exercise diminishes the glycosuria, and if there is a satisfactory response to dieting, the patient may live with care for many years. Under 40 the outlook is always unfavourable, and patients under that age who progress satisfactorily are probably suffering from some temporary cause for glycosuria or from the renal type. Under neither of these conditions would acetonuria be found. In the renal type there are no symptoms, the condition being discovered accidentally or on examination for life insurance; the blood sugar is always very low; and glycosuria is persistent, under 2 per cent., and is not diminished by dieting. As a precautionary measure such patients should not take sugar, but starch need not be prohibited, and they should be re-assured. From my own experience I am sure that such cases are not uncommon.

Modern methods of treatment have considerably improved the prognosis of diabetes. Poulton found that at Guy's Hospital, whereas formerly the mortality during the first year of the disease was 16·9 per cent. it is now only 5·4 per cent.; the average mortality-rate of all cases admitted has been reduced from

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23 per cent. to 7.7 per cent.; and whereas only 9.8 per cent. were formerly made free from sugar even for a day, now 73.5 per cent. can be rendered free for longer or shorter periods. Moreover, we have learned that coma is more generally due to faulty dieting with excess of protein and fat, than an essential part of the disease.

Is diabetes ever curable? As Garrod points out, in reported cases of cure there is seldom satisfactory proof that the reducing substance was dextrose. Some have probably been examples of a transient pancreatitis. Hyperglycæmia and lowered sugar tolerance outlast the glycosuria, and it is necessary to be sure that they have ceased for at least two years before pronouncing a case to be cured. Such cases do occur, but so rarely that we are not justified in holding out this prospect to our patients. Although they should not be discouraged, it should be made clear to them that the chances of their being able to resume a normal diet are remote. They are uninsurable lives, and their liability to death from intercurrent diseases is great.

Treatment.—Diet remains the sheet anchor, but although limitation of the carbohydrate intake plays an important part in the treatment of diabetes, it does not comprise the whole of it. Diabetes is characterized by a wasteful metabolism, and the quickest way of forcing metabolism to adopt economical lines is to cut off supplies. A proof of this is the rapid fall in the output of nitrogen as soon as no food protein is taken. It is inviting disaster suddenly to cut off the intake of carbohydrate, which normally constitutes about 70 per cent. of ordinary diet, and to increase the protein, which quickens metabolism, and the fat, which, being incompletely oxidized in the absence of carbohydrate, gives rise to toxic acids. Coma is the clinical expression of this disaster.

Formerly we were on the horns of a dilemma, for if carbohydrates were not adequately restricted the disease progressed, if they were restricted too quickly toxic symptoms might ensue. The discovery, made independently by Allen in America and Graham in London, of the value of alimentary rest has revolutionized the treatment of diabetes. The value of an occasional fast had been recognized, but it did not form part of a scheme for systematic re-education of the assimilative powers of the body.

One lesson we have had to learn from the War is that we can balance our metabolism at

a much lower level than we previously thought possible. And what may be but a passing phase for the normal individual must remain a permanent state for the diabetic. He must be permanently underfed. If he can balance his metabolism when the calorie value of his food is adequate to maintain life and a fair display of energy, the outlook is good; if he cannot acquire a balance until the intake is reduced too much for this, the outlook is bad. This is the rationale of the fasting treatment of diabetes. It is not difficult temporarily to rid the urine of sugar, but it may be difficult—nay, impossible—to keep it free when the diet is increased to anything like the level to maintain life. It will be noted that this treatment does not attack the underlying cause of the disease, nor have we as yet any means of so doing. Indeed, the disease often progresses, though usually more slowly, during the treatment.

In essentials Allen's and Graham's methods are the same, though the latter is the less drastic and, in my opinion, is the less apt to disturb the general health. I therefore give the details of this plan.

GRAHAM'S METHOD (Slightly Modified)

TWO HUNGER DAYS

Tea and coffee as desired, and 500 c.c. of bovril and broth, made without vegetables, divided into two equal portions. Water or lemonade, sweetened with saccharine, can be taken *ad lib*.

TWO VEGETABLE-AND-EGG DAYS

Breakfast.—Two scrambled eggs, with tea or coffee. 2 oz. (50 grm.) of lettuce, watercress, or tomato.

Lunch.—8 oz. bovril or broth. One poached egg on spinach. Any green vegetables, with 1½ oz. of butter. The total amount of the vegetables for the meal to be 6 to 8 oz.

Tea.—Tea or coffee, lettuce, watercress, or tomato, 50 grm. or 2 oz.

Dinner.—8 oz. bovril or broth. 2 eggs, cooked as desired—e.g. as savoury omelette. 6 to 8 oz. green vegetables, with 1½ oz. of butter. Water or lemonade as desired.

This diet has a calorie value of 1170, and a carbohydrate intake of about 10 grm.

LADDER DIET

After two vegetable-and-egg days, add 50 grm. of meat or 100 grm. of fish. This raises the calorie value to about 1300.

Two days later add 50 grm. of bacon at breakfast and omit one egg.

Add another 10 grm. of butter to the vegetables. The calorie value is now 1595.

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Two days later, add 50 grm. of sardines at lunch, and omit one egg, or if the fish has previously been given, omit this and add 100 grm. of meat. The calorie value is now 1635.

Two days later add 50 grm. of ham and omit another egg. The calorie value is now 1795. The quantities of sardines and ham may be doubled if the patient is hungry and the degree of acetoneuria is slight. This brings the calorie value up to 2145.

It will be noticed that it takes twelve days for this diet to reach the top of the ladder. If the patient is free from sugar when at the top of the ladder, add either 100 c.c. of milk (= 4 grm. carbohydrate) or 10 grm. of bread (5 grm. of carbohydrate). Increase by the same quantity every other day until the limit of carbohydrate tolerance is reached. If the patient is not sugar-free when at the top of the ladder, repeat the whole process.

In general terms, I give two consecutive vegetable-and-egg days once a fortnight, and two hunger days, followed by two vegetable-and-egg days, once a month, returning to the standard diet, as determined for the particular patient, immediately after these days. But the details of the after-treatment must depend on the individual case. Rest in bed is advisable, at any rate till the calorie value of the food reaches 2,000.

It will be noted that the more modern treatment of diabetes does not diminish our difficulties with regard to bread. As Osler says: "Most of the gluten breads are unpalatable, and the rest are frauds." I distrust all diabetic breads for which, while it is admitted that they are not carbohydrate-free, it is claimed that they can be assimilated by the diabetic. I have frequently found that such claims are unjustified, and the patient, believing that he can take such bread freely, often increases his glycosuria.

Some such breads contain very little less carbohydrate than ordinary bread, and are much more expensive. On the other hand, a bread which is really free from starch, such as Callard's Casoid (or Callard's Kalari biscuits), is very rich in protein, and on the modern plan of regulating the protein intake, this offers difficulties. An ounce of the new Casoid bread contains about 9 grm. of protein, and this must be taken into account in constructing the dietary.

On the whole, it is better to accustom the patient, if possible, to do without bread and get the required bulk of food from green

vegetables, then allowing the small amount of ordinary bread permitted by the carbohydrate tolerance, when it has been determined.

In order to afford some variety in the diet I am accustomed to give the patient a table of carbohydrate equivalents, showing the total amount of carbohydrates permitted.

TABLE OF CARBOHYDRATE EQUIVALENTS

	Weight		Grm. Carbohydrate
Bread	1 oz.	=16
Potato	1 oz.	= 8
Milk	5 oz.	= 6
Green peas	2 oz.	=10
Apples	2 oz.	=10
Artichoke	2 oz.	= 8

If, for example, the patient's carbohydrate tolerance has been determined as 50 grm. per day, allowing for the fact that the "carbohydrate-free" diet contains 10 grm. of carbohydrate, the patient is allowed to select not more than 40 grm. from any of the articles in the above table. Sudden relaxations in diet are always dangerous.

Control of acidosis.—This is now chiefly effected by so regulating the diet as to diminish the intake of protein and fat while allowing such carbohydrate as is tolerated. Many substances have been tried to determine whether they can replace carbohydrate in metabolism. Citric acid, glutaric acid, glycerin, and alcohol can all do so to some extent. Citric acid is contained in the lemonade which it is advisable for a diabetic to take, and this is generally appreciated when thirst is severe. Citrates should be added to the alkaline mixture described later, with the same object. Hirschfeld has obtained good results from glycerin. Alcohol certainly seems effective in some cases. It is both a source of energy and anti-ketonuric. The risks of the alcohol habit must be taken into consideration in each individual case, and not more than 1-2 oz. of pure spirit allowed a day. As has already been mentioned, alcoholic excess will actually excite glycosuria. Malt liquors, sweet wines, champagne and liqueurs must not be taken. In some cases a baked apple will diminish the acidosis.

The newer methods of treatment make administration of alkalis to neutralize the acids less necessary. Moreover, we place less reliance on them since we realize that even neutralized diacetates are toxic. Still, when

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the total acidity of the urine and the ammonia remain high, alkalis may be of some help.

Normally, 2 dr. of bicarbonate of soda should render the urine alkaline for twenty-four hours. If excess of acid is being formed this is not enough. In some cases it is impossible to make the urine alkaline, whatever the dose of alkali. If the condition improves under treatment, the urine will become alkaline and the amount of alkali may be gradually diminished without return of the acid reaction. It is better not to give sodium alone, as is often done, for the tissues are losing other bases as well, particularly calcium, which is an additional factor in acid intoxication. I employ the following mixture based upon the relative proportions of the metals normally present in the urine :—

R \bar{y} Sod. bicarb. \bar{z} i.
Pot. cit. gr. xxx.
Calc. carb. gr. iii.
Mag. carb. gr. iii.
Aq. ad \bar{z} i.

Half an ounce of this is given three times a day or increased to ounce doses every three hours according to the severity of the case. It does not matter if the intensity of the diacetic reaction is temporarily increased : this merely means that the acid is being excreted more readily in combination with these metals instead of with the ammonia obtained by breaking down proteins.

Drugs.—Apart from the alkaline treatment for acidosis, it cannot be said that drugs play a very useful part in the treatment. Opium and its derivatives may certainly diminish glycosuria, probably by diminishing the general metabolism. Codeia is the favourite preparation, and is given in $\frac{1}{2}$ -gr. doses three times a day, which may be increased until the patient is taking 3 or 4 gr. a day ; it is best prescribed in a pill with $\frac{1}{4}$ gr. each of extract of cascara and extract of nux vomica to diminish its constipating tendency. It should not be used as a routine, but when the glycosuria has been diminished as much as possible by diet, the fall may be continued by the use of this drug. Patients occasionally show marked intolerance of it. Salicylates sometimes definitely diminish glycosuria, but have the disadvantage of masking the iron reaction for diacetic acid. Trypsinogen, calcium iodide, sumbul, and uranium nitrate have all been found of little avail, and organotherapy has been a failure. Considering the nature of the disease, it is not to be expected that drugs would have any

real effect upon it. Tirard finds that saline aperients may diminish the sugar for a time. In general, anything which increases the digestion of starch, such as diastase, tends to increase glycosuria, by raising the blood sugar more rapidly.

Climatic and spa treatment.—Generally speaking, diabetics do better in a moderately warm climate than in a cold. A long journey is a severe tax, and should not be lightly advised. Carlsbad, Marienbad, and Neuenahr have a great reputation for the treatment of glycosuria in stout subjects. The mild régime of the Hermitage at Evian suits some cases well. and Mondariz, in Spain, has a national reputation which is likely to spread.

Treatment of complications. Diabetic coma.—On the first indication of coma it is advisable to have the rectum cleared and 3 per cent. of bicarbonate of soda with 4 per cent. of lævulose given by gravitation enema until a litre of fluid has been administered. The diet should be relaxed by the addition of two pints of milk with any other form of carbohydrate for which the patient has shown a limited tolerance. When the patient has once become comatose a temporary rally is the most that can be hoped for ; even this may be of value in order to enable him to sign a will and to recognize his friends. From the median basilic vein 10-15 oz. of blood should be removed, and then 30 oz. of normal saline containing 2 per cent. of sodium bicarbonate may be slowly infused at rather above body temperature. This may be repeated in the other arm four to six hours later. There is sometimes striking improvement, but only for a short time. Sometimes 2 per cent. of lævulose is added to the fluid, but I am not convinced of its advantages when the patient is actually comatose.

The vein should be cut down on, and no attempt made to introduce a needle into it through the skin, because of the collapsed condition of the vessel.

Diabetic neuritis.—The best treatment is to diminish the hyperglycæmia on which the neuritis depends. Aceto-salicylic acid may relieve the pains very considerably. Ionization, electrical baths, and massage are of great help when tenderness has subsided.

Perforating ulcer.—All pressure must be taken off, and the part completely rested, any sodden epithelium being removed and the ulcer washed with some antiseptic such as flavine, a saturated solution of picric acid, or equal parts

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of warm water and 10 vols. per cent. of hydrogen peroxide two or three times a day. It is then well dusted with equal parts of zinc oxide and boric acid. Later, healing may be promoted by an ointment of 5 per cent. Scarlet R. in vaselin.

Gangrene.—It will generally be found that arterio-sclerosis is present as well. Early amputation is indicated in cases of moist gangrene, as it tends to spread rapidly. There is not the same need for haste in the dry form; it spreads less rapidly, and a definite line of demarcation will appear. Before operation, local treatment should be carried out on the same lines as for perforating ulcer. However desperate the condition may appear, operation should not be refused, since the results as to life are sometimes surprisingly good.

Carbuncle should be treated on ordinary lines.

Pruritus vulvæ will subside if the glycosuria can be controlled. Frequent bathing, with subsequent applications of a lotion composed of $\frac{1}{4}$ dr. glycerin of tannic acid and of sulphurous acid in an ounce of distilled water, is the best local measure if it can be borne; it prevents fermentation in the saccharine urine which excites the pruritus. An ointment of 10 gr. of menthol in an ounce of vaselin, or a lotion of 40 min. of chloroform in an ounce of distilled water, may also help. Liquid paraffin I have found useful, while a dose of X-rays is sometimes very helpful.

W. LANGDON BROWN.

DIABETES, PHOSPHATIC (*see URINE, EXAMINATION OF*).

DIACHYLON POISONING (*see POISONS AND POISONING*)

DIAPHRAGMATIC HERNIA (*see HERNIA, ABDOMINAL*).

DIAPHRAGMATIC PLEURISY (*see PLEURISY, DIAPHRAGMATIC*).

DIARRHŒA.—Mere frequency of defæcation is not diarrhœa, for this may even be associated with constipation. The bulk of fæces excreted in twenty-four hours is generally excessive, but this again is not an essential factor, as excessive fæces are occasionally formed when the passage through the alimentary canal is not abnormally rapid. The one essential factor in diarrhœa is the abnormally rapid passage of the food-residue through the alimentary canal.

Etiology. 1. Excessive stimulation of motor

activity.—The most common cause of diarrhœa is the presence in the food of excess of the mechanical and chemical stimulants of intestinal activity. Thus over-indulgence in green vegetables, in salads, and still more frequently in fruit is a familiar cause. Chemical irritants may also be swallowed in decomposing food, as in some cases of meat- and cheese-poisoning, but food-poisoning more often causes diarrhœa by giving rise to a bacterial infection of the intestine than by the introduction of toxins already present in the food.

Diarrhœa often results from the habit of taking aperients, either in excess of what is required for the correction of chronic constipation or even when the bowels, left to themselves, would act quite normally.

Chronic diarrhœa is most commonly due to excessive fermentation of carbohydrates or putrefaction of proteins. In many cases this is a result of a definite infection with some pathogenic organism, the effect of which depends upon whether its activity causes fermentative or putrefactive changes. The infection usually gains access to the intestines in contaminated food or water. Acute diarrhœa is generally the immediate result, but it often becomes chronic, or it disappears temporarily and leads to recurrent acute attacks, which are often separated by periods of constipation. In some cases the infection is derived from pyorrhœa alveolaris or some septic focus in the nose or pharynx, and in rare cases chronic appendicitis is the source of a chronic infection of the colon. Excessive fermentation and putrefaction also result when for any reason the digestion of carbohydrates or proteins respectively is deficient. In inflammatory conditions of the intestines excess of protein, liable to undergo putrefaction, is derived from the exudation.

Diarrhœa is sometimes gastric in origin. This *gastrogenous diarrhœa* occurs whenever the gastric juice is deficient or absent. An abnormal number of organisms reach the intestines in these circumstances, as the partial protection afforded by the bactericidal action of the hydrochloric acid in the stomach is lost. As the connective tissue of meat and the cellulose of vegetables are normally softened by the hydrochloric acid of the gastric juice, undigested lumps of meat and fragments of vegetable leave the stomach and pass through the small intestine to the colon, where they are liable to undergo bacterial decomposition and so give rise to diarrhœa. The irritation of the mucous

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membrane of the intestines by the insufficiently divided fragments of food and by the products of bacterial decomposition is likely to result in secondary enterocolitis. Chemical stimulants to intestinal activity may be produced in the body and excreted into the colon; this is the cause of diarrhœa in uræmia, Graves's disease and septicæmia.

2. Over-excitability of the neuro-muscular mechanism which controls the intestinal movements. (a) *Lienteric or postprandial diarrhœa.*

—Under normal conditions the entry of food into the empty stomach gives rise to a gastro-colic reflex, which is the chief stimulus to the movements of the colon. This should only be followed by defæcation after breakfast, but in some individuals the gastro-colic reflex is abnormally active, and a formed stool, followed in the course of the next hour by one or more loose stools, is passed after breakfast. In severer cases the bowels are opened after dinner and less frequently after lunch, as well as once or more after breakfast, the stools again being often soft or fluid.

(b) *Nervous diarrhœa.*—In some patients, who are often not otherwise neurotic, attacks of diarrhœa occur whenever they are in any place where it would be awkward for them to relieve themselves; when this has once happened it is likely to occur again in similar circumstances, largely owing to fear that it will do so.

Lienteric and nervous diarrhœa are often associated together, a patient suffering from the former being particularly likely to feel an urgent desire to defæcate if he is at a dinner-party, or in a railway carriage without a lavatory. It should also be borne in mind that diarrhœa, whatever its cause may be, tends to be worse after meals, especially breakfast, and it is occasionally also influenced by nervousness; it is important therefore to exclude some other primary cause before diagnosing a case as one of pure lienteric or pure nervous diarrhœa.

(c) *Irritable colon.*—When the mucous membrane of the colon is acutely inflamed or ulcerated, any mechanical or chemical stimulant is likely to produce an excessive reflex response, so that the diarrhœa which is already present is aggravated. Apart from this, many people, who have lived in the tropics and have suffered there from dysentery, continue to be liable to diarrhœa for many years after their return to a temperate climate, owing to an irritable condition of the colon although the mucous

membrane as seen with the sigmoidoscope is quite healthy.

Symptomatology.—The chief and often the only symptom of diarrhœa is the abnormally frequent passage of abnormal stools. In severe cases discomfort or even pain is felt over the whole of the lower part of the abdomen for a short time before the bowels are opened. It may be followed by a sensation of soreness, but the abdomen is neither tender nor rigid. The passage of a large and watery stool is often followed by a feeling of exhaustion and faintness, which may be accompanied by sweating and coldness of the extremities.

In severe cases of acute diarrhœa and in persistent cases of chronic diarrhœa the nutrition suffers and the patient loses weight; sometimes an extreme degree of emaciation results. When the diarrhœa is associated with abnormal bacterial activity in the intestines, especially if this is of the putrefactive type, more or less severe symptoms of intestinal intoxication may develop.

Diagnosis.—When a patient complains of diarrhœa, it is first necessary to ascertain whether the passage of fæces through the intestines is really taking place with abnormal rapidity. The stools of every patient supposed to be suffering from diarrhœa should be examined; if they are of a uniform semi-solid or fluid consistence, true diarrhœa is probably present, whereas numerous stools, if they are small and solid, and fluid stools containing small solid fragments, suggest a diagnosis of *pseudo-diarrhœa*. In a doubtful case two or three charcoal lozenges should be given with some food immediately after the bowels have been opened in the morning; each stool is now examined, and the time which elapses before black fæces are passed is noted. If charcoal is seen in the stools before the next morning, true diarrhœa is present. In severe cases it may appear within a few hours; if in less than four hours, the small intestine must be involved as well as the colon. If no charcoal appears within forty-eight hours, constipation and not diarrhœa is present.

The most common cause of pseudo-diarrhœa is *dyschezia* (see p. 286); although the rectum is never properly emptied, the patient may feel a constant desire to open his bowels, and as a result of his efforts a very small quantity of hard fæces is passed. If, as is often the case, the constant presence of fæces in the rectum gives rise to catarrhal proctitis, more or less mucus is passed in addition. In slight cases there

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may be nothing more than a thin layer over the hard lumps of faeces; in severe cases a larger quantity of fluid mucus, which is often stained brown, is passed either alone or with hard particles of faeces. In all such cases the discovery of faeces in the rectum immediately after the bowels have been opened should remove any further doubt as to the diagnosis. A growth of the rectum or pelvic colon, and less frequently a growth in other parts of the colon, may lead to pseudo-diarrhoea, faeces being retained above the growth, while the serous and often blood-stained exudation from its surface, together with mucus from the bowel below, is passed at frequent intervals, so that the patient regards himself as suffering from diarrhoea. The character of the stool would at once make it obvious that further examination by means of the sigmoidoscope and with the X-rays after a barium meal and a barium enema is required, even if nothing abnormal is felt in the abdomen or on rectal examination.

Just as constipation may result from excessive digestion and absorption of food ("greedy colon," p. 287), so pseudo-diarrhoea may result from deficiency in the absorptive power of the colon ("anorexia of the colon"). Excessive quantities of faeces are passed, defaecation occurring with abnormal frequency, but their consistence is generally normal, as also is the rate of passage through the intestines.

When it is known that genuine diarrhoea is present, it is next necessary to determine whether it is due to a *functional* or an *organic* cause. The history is often a great help. Lienteric or nervous diarrhoea dating from childhood, especially if any other members of the patient's family are similarly affected, is almost certainly functional, unless it is due to achylia gastrica (see STOMACH, FUNCTIONAL DISORDERS OF). A very acute onset suggests that the cause is some toxic or infective agent, whereas a more gradual onset of chronic diarrhoea in a middle-aged person, whose bowels have hitherto been regular or who has actually been constipated, suggests the presence of a growth. A careful abdominal and rectal examination should be made in every case; the former may reveal the presence of a tumour or an abnormally dilated or contracted condition of the colon; by means of the latter the existence of ulcerative colitis may be recognized as well as the presence of a growth in the rectum or pelvic colon. In doubtful cases a proctoscopic and sigmoidoscopic exam-

ination should also be made before deciding that the diarrhoea is functional in origin, as the mucous membrane of the accessible part of the colon is almost always involved when diarrhoea is due to colitis.

The examination of the stools is of the greatest importance. The presence of mucus, blood, or pus indicates some form of colitis or a growth. Excess of vegetable residue is often recognizable in fermentative, and of animal residue in putrefactive, diarrhoea. The faeces are often frothy, and have an acid smell and reaction when fermentation is excessive; they may be alkaline and have a putrefactive odour when excessive putrefaction is present. A specimen of the stool made thin with water should be incubated for twenty-four hours in an apparatus in which the gas evolved can be collected. Normally little or no gas is formed, and the stool remains almost neutral in reaction. Excess of gas collects both in fermentative and putrefactive diarrhoea; in the former it is odourless and the stools are found to have become very acid, whereas in the latter the gas has a very unpleasant odour of putrefactive products and the stool is very alkaline.

In some cases a bacteriological examination should be made. A relative excess of streptococci, or the presence of abnormal organisms, such as pneumococci or dysentery or tubercle bacilli, may be discovered.

Prognosis.—If thorough treatment is instituted from the onset, the prognosis of diarrhoea is good, unless it is due to some serious organic disease. If, however, it is neglected, strict treatment requires to be continued for a prolonged period in order to be successful.

A tendency to lenteric and nervous diarrhoea often remains throughout life, though considerable improvement and even a cure can result from treatment. Unlike other forms of diarrhoea, they rarely affect the general health, and are chiefly troublesome on account of the inconvenience they cause.

Treatment.—In all cases of chronic diarrhoea recovery occurs most rapidly if the patient remains in bed during the first few days of treatment. Diarrhoea, which may have been present for months, often disappears in a few days and the patient may even become constipated. Unless, however, other treatment is instituted, the diarrhoea is very likely to return as soon as the patient gets up again. As the improvement which results from staying in bed is due in part to the rest and in part to

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warmth, it is important for the patient to avoid over-exertion and to keep his abdomen warm by means of a woollen binder for a considerable period after the symptoms have disappeared. Exposure to cold should be avoided as much as possible, and if the patient feels chilled at any time he should have a hot bath and go to bed at once.

Acute diarrhœa.—If a patient is seen within twelve hours of the onset of an attack of acute diarrhœa due to food poisoning, he should be given $\frac{1}{2}$ –1 oz. of castor oil to clear the irritant material out of the small as well as the large intestine, unless the diarrhœa is so severe that it appears probable that this has already occurred. No food should be given for twenty-four hours or even longer in severe cases, but the patient may drink as much water as he likes. Arrowroot made with water should then be given, but nothing else until the diarrhœa has ceased. Milk, junket, bread-and-butter and milk puddings are next allowed, after which a gradual return should be made to an ordinary diet, the speed with which this is done depending on the degree of severity of the case. The only drug which is of real use in acute diarrhœa is opium and its alkaloids. If the diarrhœa shows no signs of abating after twenty-four hours, some preparation of opium, morphine, or codeine should be given, the dose being regulated according to the severity of the diarrhœa and the general condition of the patient, but sufficient must be given to stop the diarrhœa within forty-eight hours.

Chronic diarrhœa.—The successful treatment of chronic diarrhœa depends upon the recognition of its cause.

1. *Excessive stimulation of the intestinal movements.*—Whatever the actual cause of the diarrhœa in this class of case it is important to avoid anything which could produce mechanical irritation of the colon. The food should be thoroughly chewed, and nothing which is difficult to break up completely, such as new bread, cheese, and tough meat, should be eaten. The patient must avoid all raw vegetables in salads and pickles, and cooked green vegetables are only allowed as purées. The pips and skins of fruit, whether raw, cooked, or in jam, must be avoided.

In *fermentative diarrhœa* all food containing carbohydrates should at first be completely excluded from the diet, with the exception of malt extract and milk. No alcoholic drinks, except a little weak whisky, are permitted.

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When the diarrhœa has stopped, cellulose-free starch, such as arrowroot, may be given, but no potatoes or white bread for several weeks, and no vegetables containing a greater quantity of cellulose until still later. Some active preparation of pancreatic ferments, such as pankreon, should be given with each meal.

A lacto-vegetarian diet is most suitable for *putrefactive diarrhœa*. Some of the milk may be replaced by milk or cream-cheese soured with a pure and active lactic-acid organism. No meat should be allowed until the diarrhœa has ceased for three or four weeks, and game and over-ripe cheese should not be eaten for many months.

Gastrogenous diarrhœa rapidly improves on the diet already described as suitable for putrefactive diarrhœa, but a relapse is certain to follow a return to an ordinary diet unless hydrochloric acid is given (*see Achylia Gastrica*, under STOMACH, FUNCTIONAL DISORDERS or).

2. *Nervous and lenteric diarrhœa.*—Nervous diarrhœa is often completely uninfluenced by diet, but drugs which diminish the activity of the gastro-colic reflex are very effective. A mixture containing bromide of potassium, 5 gr., and tincture of belladonna, 5 min., taken immediately before meals, is all that is required in mild cases; in severer cases a small dose of codeine should be added. The exact dose of each drug must be varied to suit the patient, as different individuals react very differently to these drugs, especially to belladonna. Arsenic has been recommended for lenteric diarrhœa, but I have rarely found it of any use, and in some cases it has actually aggravated the condition. When the diarrhœa has been completely controlled, the quantity of each drug should gradually be reduced; then the doses before lunch and dinner, and finally that before breakfast, can be discontinued. In most cases it is still advisable to allow the patient to have the medicine, or a pill containing belladonna and codeine, always with him so that he can take a dose before going to a dinner party, or on any other occasion when he fears that he will have diarrhœa.

For the treatment of diarrhœa secondary to colitis, *see COLITIS*.

A. F. HURST.

DIARRHŒAL DISORDERS OF INFANTS.—A satisfactory classification of that group of alimentary disorders in children which is characterized chiefly by diarrhœa and vomiting, in which morbid anatomy plays but a small

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part, and in which the rôle of micro-organisms is uncertain, is not yet possible. Different schools of thought regard the question from very different standpoints. Some believe that all but the very mildest examples are referable to infection, and diligent search has been made to inculcate particular organisms, with very variable and discouraging results. Careful bacteriological examination of the stools has demonstrated different micro-organisms—aerobic and anaerobic, proteolytic and fermentative—at various times in different areas, but their etiological significance has yet to be proved. Others blame some element in the diet and relegate infection to a position of relative unimportance. At first casein was regarded as the peccant element (Cheadle, Biedert, Meigs), next fat (Czerny, Keller), then carbohydrate, and finally the whey of cow's milk through the intermediation of carbohydrate fermentation (Finkelstein, Langstein, Meyer). A discussion of these various theories would be out of place here. No endeavour is made to adhere to any particular school of thought, but a clinical classification of the digestive disorders as they are met with in infants in this country is attempted. The subject will be considered under the following heads:—

1. ACUTE DYSPEPSIA.
2. CHRONIC DYSPEPSIA.
3. SUMMER DIARRHŒA.

1. ACUTE DYSPEPSIA OF INFANTS

Etiology.—Some error in diet is generally responsible. The milk may have been given in too great quantity, or too quickly, or irregularly. It may have been cold or stale or sour, or the mixture may have been quite unsuited to the baby's age. On the other hand, the feeding may have been unexceptionable, but the infant's powers of digestion temporarily diminished or in abeyance as the result of some acute infective disorder, some temporary disability, or some fault in infant hygiene. Persistent slight overfeeding may culminate in an attack, and is a common cause. Exposure to cold is an occasional cause. Like all the dyspeptic disorders, it is much commoner in bottle-fed infants, and, should it occur in those fed naturally, is generally mild in degree.

Symptomatology.—Vomiting is an early symptom; the vomit is sour or rancid and contains tough curds. Bile may be present and, if gastritis is produced, mucus also. When the cause is some gross error in diet, such as a

cold or sour feed, it may be followed immediately by severe gastric pain, causing the child to shriek or to be in great distress until relieved by the vomiting. In mild cases the child's discomfort may be little in evidence, and vomiting may be the sole symptom. Later, in all but mild cases, the vomiting is succeeded by diarrhœa and colic. Fluid offensive stools are passed which may be altered in colour, being sometimes green, even spinach-green, in colour, sometimes greyish and frothy; curds (casein and fat) are present, also mucus, and perhaps a little blood if actual colitis has been provoked. The abdomen may be distended from flatulence, and the child may cry from abdominal discomfort and colic, often lying with the legs drawn up over the abdomen. There may be some retraction of the head. The countenance becomes greyish and the lips tinged with blue. Some rigidity may appear and be accompanied by a state of semi-consciousness, while occasionally true convulsions supervene. Fever may be present, but it is usually slight.

Diagnosis.—The first step in diagnosis is to determine whether the condition merely complicates an *infective disease* such as an exanthem, broncho-pneumonia, or urinary-tract infections, or whether it is a primary digestive disorder. With this end in view a thorough routine examination should always be made.

Between *summer diarrhœa* and acute dyspeptic diarrhœa no hard-and-fast line can be drawn. The epidemic incidence of the former and its occurrence in the summer months may help. In summer diarrhœa the stools are often more watery (choleraic), or alternatively more often contain blood and mucus, the fever is higher, and toxæmia and prostration are more marked.

Simple regurgitation of food due to slight overfeeding must not be confused with this disorder. Without pain or effort a small amount of milk returns almost unchanged. There are no firm curds nor sour smell. Hiccoughing may be present.

Hypertrophic stenosis of the pylorus should be considered, especially when vomiting occurs in breast-fed babies. It is accompanied by infrequency of the stools instead of by diarrhœa.

Treatment.—The occurrence of the affection in breast-fed children is no sufficient reason for weaning, except in very rare instances. Any faults in the feeding with regard

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to frequency, duration, or regularity should be corrected. Scrupulous cleanliness of the breast should be enforced, and the comforter made taboo. The disturbance often arises through the baby being put to the breast hurriedly as an episode during the course of a busy day, insufficient time being allowed for proper preparation of the breast, sufficiently long nursing, or quiet and careful handling afterwards. An initial dose of castor oil (1 dr.) may be given, and thereafter all that is generally necessary is a temporary abstinence from nursing, followed by a slow return to the usual régime. This may be accomplished by missing two feeds, thus permitting a six-hours' rest to the stomach, and then breast-feeding for five minutes only at hourly intervals. As the vomiting and diarrhœa improve, the duration of the feeds and the length of the intervals may gradually be prolonged until the normal is attained. The first breast milk, being weaker in fat, is more easily digested during the earlier stages. In more severe cases and in weakly infants the breast milk may be given by a spoon at short intervals and in small amounts, and, in the case of the most enfeebled and exhausted, by a dropper in amounts as small as 20-30 drops every half-hour, allowing a five-hours' rest at night for mother and child. Only in those very exceptional cases in which milk in any form is not tolerated is artificial feeding called for. It must then consist of milk-free substitutes such as albumen water, veal tea, raw-meat juice, and vegetable broths, to which maltose and cod-liver oil may be added in small quantities to provide the necessary carbohydrate and fat.

When the infant is bottle-fed, treatment should be initiated by washing out the stomach either with plain warm water or warm water to which bicarbonate of soda (1-3 dr.) has been added. A dose of castor oil is also advisable. A longer period of starvation is required, and nothing but boiled water should be given for twenty-four hours. Thereafter the return to normal diet should be by easy stages, remembering that fat is the element which most retards digestion and is usually least well borne. Albumen water may be given for a second twenty-four hours, and if the stools are propitious this may be followed by small amounts of skimmed milk peptonized for half an hour. This may be added gradually to the albumen water or first given tentatively in one or more feeds. A dried skimmed milk, given in half strength (1-2 oz. of water) and pep-

tonized, is a suitable stepping-stone. By gradually increasing the proportion of skimmed milk and then as gradually adding peptonized whole milk, and by gradually decreasing the peptonization, a slow return is made to diet appropriate for the age. The chief mistake is to consider the lower dietary merely as a measure during the active diarrhœa and to return directly to full diet when this is over, a procedure which, more than any other, leads to chronic dyspeptic disorders and all their attendant difficulties. Warmth, quietude, and fresh air are essential.

Drugs are of secondary importance. When the stools are but little abnormal, hydrargyrum cum cretâ, $\frac{1}{2}$ gr. three times a day, may be all that is necessary. In other cases I regard a bismuth and chalk mixture as the most satisfactory:—

R̄ Bism. carb. gr. iii.
Sod. bicarb. gr. ii.
Mist. cret. ad $\overline{5}$ i.

Every four to six hours. For an infant of 3 months.

One or two grains of potassium bromide may be added with advantage if the child is restless and sleeps badly. Colic may be treated by fomentations to the abdomen or by gentle massage "clockwise" with a warmed oiled hand, while opium, either as the tincture or as pulvis cretæ aromaticus cum opio, may be added with discretion, remembering that a suitable dose for a child of one year is one minim of the tincture or one grain of the powder, and for younger infants the corresponding proportion of the dose. If the abnormal stools fail to improve in forty-eight hours, rectal lavage is often beneficial. If the child is weakly and feeble, alcohol in the form of brandy or whisky in 5-min. doses is a valuable adjuvant. Collapse will be more fully considered under Summer Diarrhœa.

2. CHRONIC DYSPEPSIA OF INFANTS

Etiology.—Chronic infantile dyspepsia may follow the acute form, but more often is gradual in its advent. More common in artificially-fed than in naturally-fed babies, it follows faults in diet and of hygiene. Though it may result from persistent underfeeding, the opposite is more often the case, and continuous overfeeding, either as a whole or with one or more of the food elements, is chiefly responsible. Great variation is met with in the physiological adaptability of different infants to cow's milk, and a proportion of any one of its essen-

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tial elements which is suitable for the majority of infants may be harmful to others. Thus some can digest and assimilate only a comparatively low percentage of fat, whilst others encounter the same difficulty with carbohydrate or with protein. Irregularity in feeding and too frequent feeds are important causes, as also are defects in the milk supplied, whether it be cold, stale, sour, or dirty. Want of fresh air, too many garments (especially in the summer months), and tightly fitting clothes are contributory causes. Sometimes the disorder dates from the first few hours of life and the injudicious use of some temporary food before the flow of milk is established, or of castor oil under the mistaken impression that meconium is harmful.

Symptomatology.—Vomiting, abnormal motions, colic, and wasting or failure to gain weight are the chief symptoms. The child becomes peevish and fretful, cries petulantly or with pain, and sleeps badly. Slight irregular fever may be present. The muscles are soft and toneless, and the skin becomes dry and harsh and loses its elasticity. There is pallor with perhaps slight blueness. The abdomen is often distended and may be tender. The normal alacrity and brightness are replaced by languor and apathy, punctuated by periods of screaming, crying, or restlessness. Development is retarded, and if the disorder continues the condition of general atrophy (*marasmus*) is produced. *Rickets* is a frequent sequel.

From the general condition and the character of the stools and vomitus some conclusion may be drawn as to the food element which is chiefly at fault. Thus, a pasty, puffy complexion and loose, green, acid stools excoriating the buttocks suggest dyspepsia from carbohydrates. With this the stools often contain curds, both those derived from fat and protein, generally mucus, and sometimes a little blood. They are emitted violently, often with a good deal of flatus, the product of fermentation. The odour of the vomit and the stools is sour. When fat is chiefly at fault a rancid odour is detectable in the breath and vomit; the stools are often bulky, grey in colour, and contain abundant fat-curds.

Large, pale, frothy, greasy, rancid stools are more characteristic of fat dyspepsia than fluid stools, though it must be remembered that faulty digestion of fat and of carbohydrates is often combined. There may be constipation instead of diarrhoea.

The stools ascribed to protein dyspepsia are

putty-like in consistence, slaty or brownish in colour, sometimes dry and crumbling, alkaline, and putrid in odour. There is too great a variation in the character of the stools and too much interaction between the processes of faulty digestion for complete reliance to be placed upon these special features, but nevertheless a careful examination of the stools gives important and valuable indication as to treatment.

Diagnosis.—The possibility of chronic dyspepsia as a complication must be borne in mind, and a systematic examination of the patient should always be made. Readily overlooked is *cystitis* or *pyelocystitis*, with which undigested motions are usually associated. The urine may be acid, turbid, and offensive both in the alimentary disorder and in the urinary infection, and diagnosis is only complete when the urine has been examined for the presence of albumin, pus, and organisms. The urinary infection is so often missed because routine examination of the urine in infants is unusual. *Celiac disease* is a digestive disorder which comes into a special category and has many particular features; it is considered under its own title. *Tuberculous peritonitis* is a rare disease of early infancy, and is distinguished by the presence of ascites, of abdominal masses, or of enlarged abdominal glands. Wasting is more rapid and more fever is recorded.

Treatment.—When the child is first seen a careful inquiry should be made into the exact diet which it has had and the times of feeding, and the milk and nursery appliances, particularly the bottle, should be scrutinized. All defects of nursery hygiene and of feeding should be put right. In mild cases, when these have been adjusted no further treatment may be necessary. A preliminary dose of castor oil is advantageous. In cases more severe, withholding of all food for twenty-four hours is advisable. During this time boiled water only should be given, except in the case of weakly infants, for whom albumen water may be allowed. Thereafter the food may gradually be amplified. The character of the motions and the previous feeding of the infant are useful guides in the selection of the diet, for they often indicate which food elements are likely to be most beneficial. Thus, if the stools show the presence of a fermentative diarrhoea due to carbohydrate dyspepsia by the features described above, a high protein content in the foods is often very satisfactory, leading rapidly to improvement. In such cases a history of

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previous feeding with an undue proportion of carbohydrates, such as obtains in most proprietary foods and in sweetened condensed milk, is often elicited. Whole citrated milk sometimes succeeds promptly, since it contains a relatively large amount of protein, which, by citration, is prevented from forming into tough curds. The citrate should be added in the proportion of 2 gr. to the ounce of milk, and the milk should be brought to the boil. It is well to begin with quite small amounts. The addition of albulactin to diluted milk is another method of increasing the percentage of protein, and in the proportion of 3 gr. to the ounce it also prevents the formation of large curds. Albumen-milk (*Eiweiss Milch*) has similar properties. To prepare it a quart of milk is first curdled with rennet and the whey is poured off. The curd is then put into a clean muslin bag and allowed to drip for two hours, after which it is found to be practically free from whey. The curd is then put into a hair sieve and gently broken up, boiled water being gradually added to make a pint of the mixture. It should be strained through the sieve often enough to provide a smooth mixture free from lumps. To this pint is added an equal quantity of previously boiled buttermilk and 1 per cent. of malt sugar. In this way a mixture containing protein 3 per cent., fat 2.5 per cent., and sugar 1.5 per cent. is obtained.

If one or other of these methods of feeding by mixtures containing a high proportion of protein fails, it is well to return to albumen-water, and thereafter gradually to add peptonized milk or a dried milk.

When the symptoms point to *fat dyspepsia* the indication clearly is to keep the fat content low. After the primary treatment by starvation and albumen water a skimmed milk may gradually be introduced. A dried skimmed or a peptonized skimmed milk will sometimes be well borne when unmodified skimmed milk fails to ameliorate the disorder. An alternative food is buttermilk. These methods should, of course, be recognized as temporary expedients; if persisted in they will cause nutritional disorders, but fat intolerance is one of the most intractable of the digestive disorders, and return to normal feeding is perforce very gradual; in many cases the fat content of the mixture will need to be kept low indefinitely.

Protein intolerance is satisfactorily met by free dilution and citration, with the addition

of lactose and cream. Top-milk mixtures serve the purpose well.

The rôle of *drugs* in chronic dyspepsia is inconspicuous. Mild cases are often benefited by *hydrargyrum cum cretâ*, to which *pulvis cretæ aromaticus* may be added if the stools are too frequent. Small doses of castor oil (5-10 min.) in an emulsion are often used in the presence of diarrhoea with watery offensive stools; to the emulsion may be added creosote ($\frac{1}{2}$ -1 min.). As a preliminary course of treatment castor oil is certainly valuable, but for protracted use I have not found it so generally satisfactory as a bismuth-and-chalk mixture. When the motions are firm and undigested, especially if mucus is present, a rhubarb-and-soda mixture is beneficial.

3. SUMMER DIARRHOEA (*syn.* Infective Diarrhoea, Zymotic Diarrhoea, Epidemic Diarrhoea)

Etiology.—Two main views are held—one that the disorder is due to infection by micro-organisms, is transmitted like other infectious diseases, and acquires epidemic incidence by the predominance of these organisms; the other that errors in the make-up of the diet are chiefly, and that organisms are only indirectly, responsible. That the disease is most prevalent in the later summer months, and is more common in hot dry summers than in those which are cool and wet, is incontestable, but the bearing of summer heat upon the etiology is a debated point, some holding that it favours infection, others that it depresses the child's powers of digestion and adaptability to foods. The arguments in favour of these opposing views cannot be considered here; suffice it to say that there is more reason to incriminate infection in this variety of alimentary disorder in infants than in either of the others, and that investigation has shown clearly that no particular organism can be regarded as specific, the intestinal flora varying in different epidemics and in different localities.

As in other infantile alimentary disorders, artificially-fed children are far more prone than those fed naturally. Dirt, overcrowding, and other insanitary states have undoubtedly a causal importance, and explain the much greater incidence among the poor, especially among the slum dwellers of large towns. Under the heading of dirt come dirty milk, dirty utensils, dirty homes, and dirty clothes. Over-clothing in the hot summer is probably a contributory factor.

It would appear that flies are capable of

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transmitting the disease, though this may only apply to particular epidemics. Weakly and premature children, those suffering from an infectious disease such as measles or syphilis, and children improperly fed or already showing evidences of digestive disorder are frequent victims. Outbreaks are to be feared in institutions where several children under the age of two years are housed together in one or more wards, especially if a liberal air-space is not available. When several children of this age are kept together, safety lies only in treating the disease as highly infectious and dealing with the stools, clothing, and bedding accordingly.

Symptomatology and varieties.—Unless there is antecedent digestive disturbance the onset is usually abrupt. The temperature rises to a variable extent, perhaps to as high as 103° or 104° F., and vomiting, colic, and diarrhoea soon make their appearance. The vomitus consists at first of sour curds, but later may contain mucus or bile, and even streaks of blood. Besides those which are alimentary, two groups of symptoms are recognizable, the one due to toxæmia, the other to loss of fluid. Owing to the relative importance of these several factors in different cases, a variable clinical picture is produced. Certain fairly definite varieties can be distinguished.

(a) **Choleraic variety** (*Cholera Infantum*, *Cholera Nostras*).—Here the initial fever, discomfort or pain, and the purging are soon accompanied by profuse fluid motions like turbid water, in which are perhaps suspended small flakes of mucus or shreds of mucous membrane—the so-called “rice-water stools.” In the severest cases collapse may occur within a few hours, the lips becoming greyish and blue, the extremities cold, the temperature subnormal, and the pulse feeble and quick. Death may soon supervene. In cases less fulminant, loss of fluid soon becomes evident and more completely dominates the picture than does toxæmia, which may be little in evidence, probably because of the free elimination. The tongue becomes dry and glazed or may be coated, and the child suffers from great thirst, swallowing ravenously any fluid which is given to it. The eyes sink in and the fontanelles become depressed, while the skin loses its elasticity and hangs in folds about the buttocks and inner sides of the thighs. Great restlessness is evident, the child’s eyes wandering incessantly and the limbs moving continually. The abdomen is generally retracted,

and through the shrunken abdominal wall the “ladder pattern” of actively contracting bowel can often be seen. The urine is diminished in amount and may contain albumin and sometimes casts. Occasionally suppression occurs. Fatal collapse is to be feared. In its absence some fever may persist until the termination of the attack, when it gradually subsides by lysis. Throughout the attack, fluid bile-stained vomiting may amplify the fluid loss, and colic and loss of sleep may intensify the exhaustion.

(b) **Dysenteric variety** (*Ileo-colitis*).—Less common in this country than the choleraic variety, dysenteric diarrhoea would appear to be far more prevalent in the United States and Eastern Europe than here. Organisms of the Shiga-Flexner group are probably responsible. In contradistinction to the choleraic form, which possesses no gross morbid anatomy, this variety is pathologically an ileo-colitis, and inflammatory changes, together with ulceration, are found in the bowel, especially the lowest third of the ileum and the colon. In London, in my experience, cases of this kind are most often found towards the end of the diarrhoeal season.

In the main the symptoms are similar to those of summer diarrhoea generally, but certain distinctive features are worthy of note. The fever is usually high, and the divergence between the rectal and axillary or oral temperatures is considerable. The stools are not of the “rice-water” kind, but loose, undigested, and offensive, and contain much mucus and often blood. Blood and mucus may be evacuated independently of the stools. The course may be protracted into several weeks. Toxæmia as shown by fever, torpor, and prostration are more in evidence than symptoms of dehydration. The abdomen is usually distended and tender, and pain is generally prominent.

(c) **Toxic variety.**—Here the toxæmia predominates. Though the stools are abnormal and undigested there may be little or no diarrhoea, and the same is true of the vomiting. The child, however, is obviously poisoned. In the gravest cases the temperature may be depressed or normal, in those less severe it *seems* high. The clinical picture suggests typhoid fever, the tongue being coated, the breath foul, the abdomen tumid, the child prostrate and semiconscious. Restlessness is evident. *Apathy* and torpor may deepen into coma, the *main* cause of death, or twitching and *convulsions* may supervene. The pulse is usually *weak*.

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irregular, and feeble, while the respirations are variable, being sometimes quick and shallow, sometimes deep and laboured, and often irregular and broken by periods of apnoea in the later stages. Acetone is frequently detectable in the breath. The condition is very serious and often fatal. The onset of diarrhoea is a favourable sign.

Though these groups can be defined with fair precision, the symptoms of summer diarrhoea are very variable, and many cases are met with which cannot be included in either group. Some are indistinguishable from ordinary dyspeptic diarrhoea; in others neither the character of the stools nor the degree of toxæmia is a particular feature.

Complications.—*Collapse* is a serious complication, though by no means hopeless, for it may recur on several occasions in cases which recover if treatment be energetic. Among common respiratory complications are *bronchitis*, and *congestion and collapse of the pulmonary bases*. Râles and weak breath-sounds at the bases with diminished resonance may almost be said to be the rule in the severer cases. *Broncho-pneumonia* of a low and irregular form is not uncommon and is a very fatal complication. Two developments of serious import are *superficial œdema* and *purpura*, especially the latter. The œdema is often confined to the backs of the hands and dorsa of the feet; the purpura is of very grave omen, and is often confined to the abdominal wall, though it may be more general. The points of hæmorrhage are often crowded together, producing a dusky purplish though speckled area on the abdominal wall; in its presence recovery is almost though not entirely hopeless. *Nephritis* is often found after death, and may cause urinary changes during life. Should *suppression* supervene the chances of recovery are small. Many cases of summer diarrhoea fail to recover completely, but, the acute symptoms being over, *intractable and persistent dyspepsia and malnutrition* (decomposition) continue, probably due to failure of absorption and of metabolic activity of the tissue cells.

Morbid anatomy.—Local changes are very slight, except in the dysenteric form, in which inflammatory changes and ulceration may be found in the ileum and colon. Usually the bowel wall is thin and translucent, with perhaps a slight excess of adhering mucus and a few hæmorrhages. The same may be said of the stomach, which in addition may contain

a little altered blood. The lymphatic tissue generally is atrophied, including that of the bowel, and the thymus is markedly shrunken. Parenchymatous degeneration of the kidneys and liver is found.

Treatment.—The primary object of treatment is not to stop the diarrhoea, but to assist in the elimination of the toxins and at the same time to conserve the patient's strength. Warmth and fresh air are essential. The child should be placed between blankets in a warmed cot in an airy room or on a balcony, and should be handled as little as possible. A relay of hot-water bottles of small volume is better than one large one refilled occasionally; medicine bottles wrapped in flannel will do. When practicable an electric-light bulb suspended from a cradle underneath the bedclothes is an excellent device for promoting continuous warmth. Soiled napkins should be placed in antiseptic solution as soon as removed, and the greatest care taken to avoid contamination of the nurse's hands or of feeding utensils. When the case is seen early an eliminative dose of castor oil or calomel should be administered, whilst elimination may also be assisted by washing out the bowel and stomach; in the presence of profuse diarrhoea or vomiting, lavage is, of course, unnecessary. Milk should immediately be withheld, and for twenty-four hours only boiled water given (*ad libitum*); this may be succeeded by albumen-water, which should be continued until some improvement accrues. At this stage the most useful drug is castor oil in 10- or 5-min. doses, which may be given in a suitable emulsion every four hours. A suitable prescription is:

R̄ Ol. ricin. Mv.
Glycer. q.s.
Aq. menth. pip. ad ʒi.

The value of *normal saline* is well attested; it assists elimination, dilutes the toxin, and replaces fluid loss, as well as combating collapse and suppression of urine. The saline may be given hypodermically, about 4 oz. at a temperature of 100° F. being injected into each axilla or into each flank. With profuse diarrhoea the rectal route is impracticable, while the intraperitoneal method, advocated by some, has no advantages commensurate with the added risk. Saline is especially indicated in choleraic cases and in threatened or present collapse. In such cases it may be given continuously through a narrow-bored needle, from a vacuum flask, the two being connected by rubber

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tubing, and precaution being taken to watch the local effect lest too great tension lead to sloughing of the skin around the site of injection. The hypertonic saline treatment, advocated by Sir Leonard Rogers for cholera (q.v.) is no doubt advantageous, but hard of attainment because of the small size of the veins.

When *collapse* is threatened, stimulants are called for. The best of these is brandy, 10-15 min. every three or four hours, in a little warm water. Camphor in olive oil, 5 min. of a 1:15 or 1:30 solution, may be injected subcutaneously with advantage. Other measures are salines as mentioned above, hot packs, and mustard baths. Neither of these measures should be withheld when collapse is actually in being.

Though diarrhoea moderate in degree needs no special treatment, it may become dangerous when profuse and repeated as in cholera infantum. The castor-oil mixture may then be replaced by a bismuth-and-chalk mixture to which opium in the form of a Dover's powder, tincture of opium, or chlorodyne may be added in intractable cases, provided that the tongue is clean and there is neither suppression of urine nor serious toxæmia. Occasionally, when vomiting and diarrhoea are profuse in the choleraic form, a hypodermic injection of morphia ($\frac{1}{8}$ gr. for a child of one year) is directly useful. It should only be given in the absence of the contraindications for opium mentioned above.

With amelioration of the symptoms, return may be made very gradually to normal feeding. A dried skimmed milk may first be tried, freely diluted, and gradually augmented by a dried whole milk or milk fully peptonized (for an hour). Thereafter diminishing peptonization and greater strengths may lead by easy stages to a normal diet. Any recrudescence of symptoms should be followed by the abandonment of milk entirely for the time.

FREDERICK LANGMEAD.

DIATHERMY.—In diathermy heat is not applied to the surface of the body, as was done in the older methods, and then conducted through the tissues, but is actually generated in the tissues themselves owing to the resistance they offer to the passage of an electric current, just as a protecting fuse is heated to the melting-point, or as the filament of an electric lamp is heated to a state of incandescence.

If the white of an egg be placed in a glass vessel of such shape that the albumen forms two lakes with a narrow canal joining them, and the electrodes of the diathermy apparatus be placed one in each lake and the current be turned on, we find that the albumen forming the canal is first coagulated; if the current be then turned off, the electrodes are found to be cool, as also is the albumen immediately surrounding them, or at any rate not warm enough to coagulate the albumen forming the lakes, although that forming the canal is cooked.

"Diathermy" is not perhaps the best name for the process, although it is the generally accepted term. Diathermancy in the case of heat is equivalent to transparency in the case of light; it refers to transmission of heat. Similarly, athermancy is equivalent to opacity in the case of light. In so-called diathermy we have to do with the transmission of electric current, not of heat; the heat is endogenous. "Endothermy" would seem to describe the process better.

In using diathermic high-frequency currents it is necessary to bear in mind that the maximum heating takes place at the smallest cross-section of the body under treatment, for here the current lines are crowded together, or, in other words, the current density is greatest. This is very noticeable when the current is passed through the body from one hand to the other, the whole of the hand being in good contact; a feeling of heat will be felt at the wrists, which present the smallest sectional area through which the whole current passes. If one hand be touching the electrode by means of one finger only, the heat is greatest in that finger and would soon become intolerable, the density of the current being so great here that coagulation or burning would soon be brought about.

These currents produce no ionic action or electrolysis, and the skin offers about the same resistance as the other tissues. Thus they differ essentially from the galvanic current.

The resistance of the human body to them is neither an entrance resistance nor a resistance of structure.

In diathermy there are two methods of treatment, the *medical* and the *surgical*.

1. In the *medical* method small heating effects are obtained with a local hyperæmia and a local elevation of temperature, which persists for some time after the application. Diathermy may be used in this way in a variety of cases.

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tions. It has a marked power of soothing pain in neuralgia, lumbago, brachial neuritis, sciatica, etc. With large electrodes a considerable portion of the body, e.g. the chest, heart, liver, or knee-joint, may be warmed through. Pain in a joint affected with gout may be relieved, the gouty deposits redissolving owing to the increase of temperature produced. The heart's action may be considerably modified in frequency and force by using two large electrodes, one in front and the other behind the chest.

2. The **surgical** method enables many operations to be performed without loss of blood and with less risk of disseminating infection, the mass dealt with being coagulated before being removed. By using a very small metal plate or a needle, the current may be concentrated on to a small area of tissue so that it can be burnt, and with the needle deep incisions may be made without hæmorrhage. The current strength can be so adjusted that the needle will cut through the tissues without causing necrosis in the surrounding structures, or it may be increased so that the tissues become charred to the depth of $\frac{1}{4}$ mm. or $\frac{1}{2}$ mm. By this method individual hair-follicles, small surface growths, warts, etc., may be destroyed, or incisions made in dealing with larger growths. Again, by graduating the density of the current, using an active electrode of somewhat larger size, a growth, e.g. in the tongue or tonsil or bladder, may be coagulated, and subsequently thrown off. I have used this method successfully in dealing with piles, also with the part of a uterine cervical growth which was projecting into the vagina. For the latter, suitable electrodes with long handles are made which can be used through a glass vaginal speculum.

Douglas Harmer and Lewis Jones have reported several cases of epithelioma of the tongue, soft palate and tonsils which were dealt with by diathermy with gratifying results. The former has also treated cases of nævi and innocent growths in the mouth and palate, and there has been no hæmorrhage. He describes his technique as follows: "A general anæsthetic is required. The growth must be sponged so that it is quite dry. The electrode or needle is plunged into the growth and the current is turned on for 3-5 seconds, by which time the neighbouring tissues are dead. A series of punctures is made so that every part of the growth is attacked; this can be done in about five minutes. There should be no bleeding even with vascular tumours." Em-

ploying a large indifferent electrode for one pole and a small active electrode for the other, an intense superficial action of small depth can be produced by using a strong current for a short time. If, on the other hand, a weaker current is applied for a considerable time, deeper action will be obtained. No action is apparent at the indifferent electrode. In this way a patch of superficial lupus may be attacked, as also may all forms of tuberculosis of mucous membrane or of skin. Using an active electrode of 1 cm. diameter and a current of 1,500 ma. for ten seconds at each application, a white spot will be found under the electrode. Using two 1 cm. electrodes applied 2 cm. from each other, strips of coagulation will be produced, and thus the area to be treated will be got over more rapidly.

In treating a disseminated lupus with small scattered foci in scar tissue or in healthy skin, smaller electrodes may be used with a current intensity of 1,000 ma. for four or five seconds. A still smaller electrode or needle may be employed for minute nodules, using 200 ma. to 300 ma. for one second. Affections of the nose and throat may be treated in this way, local anæsthesia being induced by cocaine. Diseases of the larynx are reported to have been so treated without any indication of pain, two-minute applications being given with satisfactory results; the lesion in each case is reported to have remained apparently healed. No œdema of the larynx or glottis is said to have occurred. Warty growths may be attacked with a needle electrode, causing central coagulation. Chronic ulcers may be made to heal rapidly.

A cancerous nodule in the tongue may be destroyed, using two electrodes and coagulating the growth between them. The same effect may be produced in other sessile growths by using an indifferent electrode and a small active electrode, coagulating into the nearest layers of healthy tissues beneath at once, or coagulating the superficial layers of the growth, removing them with a swab of gauze or a blunt curette, and proceeding to coagulate and remove further layers until a sufficient depth has been reached.

Diathermy may be used to arrest disease of bone. The organic elements of the bone can be coagulated and the bone then chiselled away till the coagulation limit is reached.

Hæmorrhage from a spouting artery or a large vein may be arrested by introducing a small electrode, which in a few seconds will

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produce coagulation. If necessary, an artery laid bare in a wound may be divided and the two ends coagulated by touching the wall of the vessel lightly with a small electrode and using a current of 1,000–1,500 ma., complete obliteration being secured without danger of recurrent hæmorrhage.

E. S. WORRALL.

DIET (*see* INFANT FEEDING).

DIETL'S CRISES (*see* VISCEROPTOSIS).

DIGESTIVE DISORDERS OF INFANTS (*see* DIARRHOEAL DISORDERS OF INFANTS).

DILATATION (*see* under individual organs).

DIONIUM POISONING (*see* POISONS AND POISONING).

DIPHTHERIA.—An inflammatory affection of certain mucous membranes, most frequently those of the nasal passages, fauces, larynx, and trachea, but occasionally also of others and even of the skin; it is usually characterized by the local formation of a false membrane and by peculiar toxic symptoms, and is caused by a specific micro-organism, the diphtheria bacillus.

Etiology.—Although the essential cause of the disease is the bacillus, without which there can be no diphtheria, yet there are several factors which favour either the pathogenic action of the micro-organism or its dissemination. They are the following:

Age.—The disease is met with mostly in children, especially those from 1 to 4 years of age.

Season.—In this country more persons fall with diphtheria during the end of the summer, the autumn, and the beginning of the winter than at any other season. September, October, and November are the months of its greatest prevalence. Its virulence varies considerably from time to time, and is not necessarily highest when it is most prevalent.

Climatic conditions.—Diphtheria is met with mostly in temperate climates. Its prevalence, as measured by the number of the recorded fatal cases, is higher in years of drought than in those in which the rainfall is above the average.

Dissemination.—The disease is spread either directly, as in the act of kissing, or indirectly, by infected articles, especially those used for eating and drinking. The habits of children attending elementary schools are often such

as would favour its spread (e.g. licking slates to clean them, sucking pencils, these articles being used indiscriminately by the children without being cleaned); and there is evidence to show that the aggregation of large numbers of children in schools is a contributory cause of the dissemination and prevalence of the disease.

There are reasons for believing that persons who themselves remain unaffected may acquire the bacillus in their throats from those who are suffering from diphtheria, and, acting as *carriers*, may then convey it to susceptible persons, who become infected with the disease. Another mode of dissemination is by the infection of a milk supply, either directly from a person suffering from diphtheria, or indirectly through the cow. Milch cows not infrequently suffer from ulceration of the udder; and it has been suggested that the ulcers become infected with the specific bacilli from a human source, and that the bacilli get into the milk without giving rise to any disease in the animal, which thus acts as a "carrier." Animals are very rarely the subjects of diphtheria as it occurs in the human being. Faucial diphtheria of the cat and nasal diphtheria of the horse have been described.

Pathology and morbid anatomy.—Diphtheria is in the first instance a local disease. In many cases it remains a local disease. In others, however, the poison which is produced by the diphtheria bacilli in the local exudate is absorbed into the system and gives rise to changes in certain of the tissues, these changes being manifested by appropriate symptoms. Thus, diphtheria is a local disease which may or may not be followed by toxæmia. Further, the site of the local lesion will determine the symptoms. In laryngeal diphtheria, for instance, the prominent symptoms are those of obstruction of the air-passages.

Diphtheritic membrane is at first white, or yellowish white. In those places where it is more or less intimately adherent to the subjacent mucous membrane it becomes of a darker colour as its formation progresses, and may then be of a grey or blackish hue. But where it is loosely attached it usually remains white to the end. It is often tough; but not infrequently it is of a more or less pultaceous consistency and does not come away as a definite membrane. Often it has a gelatinous appearance. It is usually fairly closely adherent to the mucous membrane of the tonsils, pharynx, palate, and vagina, so that it is removed only with some difficulty. On the other hand,

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in the nasal passages, larynx, trachea, and bronchi, and on the conjunctiva, it lies loosely upon the surface. In the case of the respiratory passages this is shown by the ease with which it is expelled by coughing or removed by instruments in tracheotomy. Consequently, when only these surfaces are involved, toxæmia is slight or absent.

When examined with a microscope the false membrane presents in some cases a granular, in others, and especially in membrane from the air-passages, a fibrillated appearance. A few leucocytes and cocci may be seen throughout the whole thickness of the membrane, but the specific bacilli are found only on the surface, where they occur in masses.

In most cases, little can be found by the unaided eye at a post-mortem examination beyond what can be observed during life. Indeed, if the exudate has cleared off before death, the appearance of the affected mucous membrane may be little if at all altered from that of health. Superficial necrosis may be found, but marked ulceration is rare. In hæmorrhagic cases, besides cutaneous and subcutaneous hæmorrhages, there may be effusion of blood into the retropharyngeal and peritoneal tissues, into the submucous tissue of the stomach, beneath the pleura and pericardium, and even into the lungs and heart muscle. There may be fatty changes in the heart, liver, and kidneys, though renal changes are rarely so distinct as to be visible to the unaided eye. In tracheal and bronchial cases there is frequently extensive broncho-pneumonia, especially when tracheotomy has been performed. The lesions which are found in diphtheritic paralysis will be described when that complication is dealt with.

Incubation period.—This may last from twenty-four hours to four days; occasionally it is longer.

Symptomatology.—The symptoms may be divided into two groups. In the one may be placed those which are due to the local action of the inflammation, in the other those which are set up by the secondary toxæmia. The latter are the same, except as regards variations in intensity, whatever the site of the local inflammation may be; the former depend upon the locality.

The mucous membranes most frequently affected are those of the fauces, larynx, trachea, and bronchi, and of the nasal passages. Much less frequently are the genital organs and the eye invaded.

Faucial diphtheria.—The attack usually begins insidiously with slight sore throat, very often so slight that a child will not complain of it. In many cases a well-defined plaque of membrane is found covering one of the tonsils on the first inspection; but if the opportunity has been given at the very earliest stage, a thin membranous film, very limited in area, will be found on one of the tonsils, the uvula, the pharynx, and occasionally the soft palate. The thin film may spread and increase in thickness very rapidly, even in the course of a few hours, so as to cover the whole tonsil. I have known, in not a few cases, a thick, tough sheet of membrane, so extensive as to cover the whole of the faucial arch, to form within twenty-four hours; but usually two or three days are required for an extension so wide. There is seldom much swelling of the subjacent mucosa, so that, unless the faucial aperture is blocked with membrane, there is little, if any, difficulty in swallowing and speaking. Very rarely does the formation of membrane go much beyond the tonsils, arch of the palate, and soft palate, so that the hard palate, the inside of the cheeks, the floor of the mouth, and the tongue are very exceptionally affected. After a few days the membrane begins to decompose and separate. It becomes of a dirty yellow, greyish, or reddish-grey colour. When not intimately adherent to the surface underneath, it comes off *en masse*, leaving the mucous membrane superficially necrosed and ready to bleed at the slightest touch; but when it is closely adherent it slowly melts away. In cases not treated with the specific serum the process of separation will take from one to three weeks. A very offensive and characteristic odour is given off from the decomposing membrane.

Soon after the formation of the membrane the cervical glands on the affected side become moderately enlarged and slightly tender. In cases in which the membrane is thick and extensive the neck becomes swollen; the skin is puffy and soft, but there is very rarely the hard, brawny, induration that is seen in the cervical cellulitis of scarlet fever and Ludwig's angina, nor are the glands matted together.

The toxic symptoms are usually in proportion to the extent of the false membrane, due regard being had to the age of the patient. They appear more rapidly and are more evident in children than in adults. At the onset there is a rise of temperature to 100°–103° F.; the pulse-rate is accelerated, sometimes to as much

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as 200 a minute; and the blood-pressure falls sharply, so that the pulse is very soft. The temperature does not remain raised for more than a few hours; it then falls considerably, even to normal, but may rise again irregularly during the course of the disease. When the membrane begins to disappear, it becomes normal and remains so, except in fatal cases, when it is often subnormal and may drop to as low as 96° or 95° F. for a day or two before death. As the disease progresses the pulse-rate usually returns to normal, but it is prone to be temporarily increased in frequency by slight causes. In fatal cases it may be irregular and infrequent. The rate may fall to 50 or 40 in the minute. Other signs of toxæmia are albuminuria, diminution in the amount of urine, vomiting, and prostration. Albuminuria usually makes its appearance about the fourth day and continues in severe cases for several weeks; the quantity of albumin is rarely great. Hæmaturia is uncommon. In most cases of any severity the amount of urine is considerably diminished; in fatal cases it may drop to 3 or 4 oz. or less in the twenty-four hours, and there may be complete suppression for two or three days before death.

Vomiting is occasionally an early sign, but usually does not occur till after three or four days' illness. In serious cases it is frequent and takes place quite independently of food.

In severe toxæmia prostration is the prominent feature. Often there is restlessness, and occasionally delirium, which, however, is rarely violent. On the other hand the patient may be very quiet and apathetic, his face without expression, pallid, and waxy, and his mental condition not infrequently quite normal, so that to the unpractised eye he may not appear to be in danger.

In the gravest cases there is some enlargement of the liver, and a blotchy, erythematous rash may come out, especially on the extensor aspects of the limbs about the joints. Hæmorrhages may occur into and beneath the skin and from some of the mucous membranes; the cutaneous hæmorrhages being small and of a purple colour, the subcutaneous larger and of a bluish tinge. These hæmorrhages are not usually numerous. Slight traumatic lesions, such as that caused by the needle of an injection syringe, are prone in these cases to set up local hæmorrhage. There may also be bleeding from the nose, gums, and pharynx,

and the patient may vomit and cough up blood, but hæmaturia is rare.

From toxæmia death usually takes place in nine or ten days, and is due to slowly progressive cardiac failure.

Laryngeal, tracheal, and bronchial diphtheria.—In most cases the air-passages are invaded from the fauces, the disease progressing from above downwards; but occasionally diphtheria commences in the larynx. In very rare cases it may, as it were, leap from the fauces to the trachea, leaving the larynx unaffected; or it may commence in the trachea. Implication of the larynx usually takes place within a week or so from the onset of the disease in the fauces. It is met with chiefly in children under 10, and is rare in adults.

The symptoms of laryngeal diphtheria are almost entirely due to obstruction of the air-passages, and in pure laryngeal and tracheal infection toxæmia is very slight or absent. The cardinal signs of laryngeal obstruction are four: loss of voice, inspiratory stridor, a harsh and frequent cough, and recession of the epigastric region, the lower parts of the chest, and of the space just above the sternum, during inspiration. Besides these there may be exaggerated action of the respiratory muscles, even of those not usually employed, cyanosis, restlessness, and an intermittent pulse. When the obstruction is complete, or almost so, the patient will, if unrelieved, die of slow suffocation.

These symptoms vary in intensity in different cases, and one is often more prominent than another. Often, too, during the progress of the disease the symptoms are enhanced by spasmodic attacks, and during such an attack surgical interference may be necessary. In other cases there are no such exacerbations, but the symptoms gradually grow worse. In very severe toxic faucial diphtheria the larynx and trachea may be involved without the manifestation of any of the signs mentioned above, and the condition may be discovered only at a post-mortem examination.

In adults the invasion of the larynx does not give rise to those urgent symptoms of obstruction which are to be seen in children. Often the only sign of such involvement is aphonia, and it is not till the bronchial tubes are affected that symptoms of dyspnoea show themselves. Hence loss of voice should always be looked upon with apprehension in a case of diphtheria in an adult.

Nasal diphtheria.—The chief symptom is a

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discharge from the nose, at first thin and watery, later muco-purulent. Not infrequently it is bloodstained. Membranous casts of one or both nasal fossæ may be shed. The constitutional symptoms are usually very slight.

Nasal often accompanies faucial diphtheria, especially in the most severe cases, the nasal passages apparently being invaded from the fauces.

Vulval diphtheria.—This is usually met with in little girls, and is uncommon in adults. It may occur alone, but more often follows the faucial form. Doubtless the genital organs are infected by the action of the patient in scratching the parts with contaminated fingers. One or both sides may be affected. There are swelling and redness of the labium majus, the inner surface of which will be found covered with ashy-grey membrane. The lesser labium may also be affected. The lymphatic glands in the groin are enlarged and tender. Toxæmic symptoms are usually present and may be severe.

Preputial diphtheria.—Membrane forms on the mucous surface, especially where it is reflected on to the glans; there is œdema of the prepuce. This form of the disease is very uncommon.

Conjunctival diphtheria.—A thin membrane lines the inner surface of the eyelid. Usually only the lower lid is affected, but both lids of each eye may be involved. There is swelling, usually moderate, of the lid. The membrane may spread on to the eyeball, in which case there are intense conjunctivitis and keratitis. Ulceration of the cornea may result, with perforation and panophthalmitis.

When there is much swelling with brawny induration of the lid, and intense conjunctivitis with a profuse purulent discharge, the diphtheria bacillus is seldom the sole cause of the inflammation.

Cutaneous diphtheria.—Diphtheritic membrane may form on the surface of a wound or on a patch of weeping eczema. If the area involved is extensive, constitutional symptoms may be present. The aseptic and antiseptic treatment of wounds has apparently rendered this variety of diphtheria much less frequent than it was in former days.

Cutaneous gangrene is occasionally set up by the diphtheria bacillus. It is probable that inoculation occurs, which is followed by inflammation and necrosis of the skin and subcutaneous tissue, and usually by considerable inflammation around the gangrenous patch.

The constitutional symptoms may be severe. I have seen one case in which death occurred from late paralysis.

In rare cases multiple infection of the skin takes place, resulting in the formation of a number of vesicles and pustules, which are slow to yield to treatment other than that by the antitoxic serum. In the few recorded cases the condition has been secondary to diphtheria of a mucous membrane.

Instances of diphtheria of the *œsophagus*, *stomach*, *middle ear*, and *external auditory meatus* have been recorded, but are very rare.

Whilst in the majority of cases of diphtheria the exudate is distinctly membranous, in not a few it is merely pultaceous; the colour is then yellowish-white rather than grey. It is especially on the fauces and on the genital organs that the exudate assumes this character.

Complications and sequelæ.—Excepting **paralysis**, these are not frequent, and this is by far the most important sequel. In the vast majority of cases there is a distinct interval between the disappearance of the local exudate with the accompanying toxæmic symptoms and the onset of paralysis. This interval may be of any duration, from a few days to a few weeks. It is commonly from two to four weeks; but in several cases I have known it to be six to seven weeks, and in one case about eleven weeks. As often as not the patient appears during the interval to have quite recovered from the attack of diphtheria. In a few very severe cases paralysis sets in as early as the fourth or fifth day of the disease, before the false membrane has begun to disappear. The more pronounced the toxic symptoms, the more likely is paralysis to occur and to be serious. Severity of toxæmia depends upon the extent and duration of the local exudate and the closeness with which it is connected with the mucous membrane upon which it rests. Hence paralysis is seen more often after faucial than after purely nasal or laryngeal diphtheria, and its incidence varies with the severity of the cases which constitute the epidemic. It is much more often met with in children than in adults, and the younger the child the greater is its liability to this sequel.

Paralysis may be expected in about 15 per cent. of all cases. It is preferable, however, to state the incidence of this sequel on those patients who survive the stage of the disease, since not a few of the cases which are fatal during this stage are of such a nature

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as almost certainly to develop paralysis were they to survive for a longer period. On this basis the incidence becomes about 20 per cent.

Paralysis nearly always shows itself first in the soft palate. The patient speaks with a nasal twang, or fluids regurgitate through his nose when he drinks. There may also be some difficulty in swallowing; attempts at drinking provoke spluttering and choking. Next follow squint and a weakness of the ciliary muscles which prevents the clear vision of near objects. The strabismus is usually due to paralysis of the left external rectus. Only in very severe cases are other ocular muscles affected, and complete paralysis of all the muscles is extremely rare. Soon the lower extremities become affected, and the patient staggers in his gait or is unable to stand. Then follows paralysis of the neck, back, and upper limbs. Lastly, the abdominal and respiratory muscles, including those of the larynx, are affected. If this occur, usually all the intrinsic laryngeal muscles are concerned, and there are present aphonia and a toneless, ineffectual cough. In rare cases the abductors alone are paralysed, and there are inspiratory stridor, recession of the chest-walls, and cyanosis. If the paralysis extends to all the groups of muscles which have been mentioned, the patient lies quite helpless. In such the facial muscles may be involved, though only to a slight extent. The sphincters or the tongue are seldom implicated.

In a case in which the paralysis is widely spread the process of generalization usually occupies a week or two, though it may take longer, or three or four days only. The latter is especially likely to occur when the paralysis has begun early.

Though paralysis nearly always starts in the palate, it may begin in any other group of muscles, and may be confined to them, so that a squint, or an inability to see near objects clearly, or an ataxic gait, may be the first and only sign. In rare cases the palsy may commence in the diaphragm and the patient may die of inability to breathe before another group of muscles is invaded.

Loss of muscular power is seldom complete; it is usually partial—paresis rather than a paralysis. When the patient is lying in bed he may be able to move his limbs quite freely, but when resistance is offered to the action of the muscles, it will be found that the loss of power is pronounced. In consequence, however, of the heart affection which is often pre-

sent in these cases it is most unjustifiable to submit a person suffering from extensive diphtheritic paralysis to a too searching physical examination.

There is loss of tactile sensation, but those of heat and cold and of pain are seldom interfered with and the special senses also escape. Loss of sensation is always partial, and is not often met with save in severe generalized paralysis. It is most marked in the distal portions of the extremities, the tip of the tongue, and the nose. Occasionally an adult will experience a numbness of the tongue as the first sign of paralysis.

In severe cases the muscles become flabby and wasted. The reactions to both forms of electricity are diminished; reaction of degeneration is uncommon. The superficial reflexes are unaltered except in the gravest cases, when they are diminished and even lost. The tendon phenomena are often absent. If the toxæmia be severe the knee-jerk nearly always disappears early, and may be absent for weeks or months, quite apart from the presence of definite signs of paralysis. Sometimes the disappearance of the knee-jerk is preceded by a stage in which it is exaggerated. The reactions of the pupils to light and accommodation are sluggish in very severe cases. Optic neuritis is rare.

Patients who are the subjects of paralysis are prone to be affected with the cardiac complications to be mentioned presently. Another troublesome and sometimes dangerous symptom is repeated vomiting. It may seriously jeopardize the patient's chance of recovery by preventing him from retaining sufficient nourishment. Attacks of respiratory distress, "*respiratory crises*," have been described as occurring during the course of paralysis, but in my experience are extremely rare. A sudden attack of dyspnoea in a patient who has paralysis of his pharyngeal and laryngeal muscles should always arouse suspicion of the escape of saliva or food into the trachea and bronchi. In two or three cases I have noted a paroxysmal secretion of saliva.

Morbid anatomy of diphtheritic paralysis.—The paralysis following diphtheria has been attributed by most observers to a peripheral neuritis. In my opinion this can hardly be the case, since there is no pain whatever along the affected nerves. The microscope reveals a breaking up and disappearance of the white substance of Schwann, followed by a similar change in the axis-cylinder. The

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follows secondary degeneration of the nerves and of the muscles which they supply. These changes are found mostly in the peripheral nerves, and not all the fibres are affected; hence the paralysis is rarely complete.

There is some evidence that the central nervous system is involved before the peripheral. Changes have been observed in cases which have been fatal at an early stage, in the cells of the anterior cornua and of the nucleus ambiguus of the vagus, but they would seem to be neither extensive nor lasting. Clinically a case of diphtheria rarely presents symptoms that suggest a gross central lesion.

Cardiac complications.—In severe cases of toxic diphtheria the toxin affects the heart-muscle quite early in the disease, and cardiac failure, sudden or progressive, is the most common mode of death. The action of the heart may be deranged greatly during the acute stage. In a few cases a dilatation of the organ takes place which may be very acute, the patient suffering from sudden and agonizing pain in the cardiac region, the heart's action becoming very frequent and irregular, and death occurring in a few hours. It cannot be too strongly emphasized that the risk of cardiac trouble is by no means over when the acute symptoms of toxæmia have passed away. The most common disturbance is irregularity and undue frequency, with or without dilatation; and this is more likely to arise if too much work is put upon the heart, even by so little additional exertion as would be necessitated by the patient's getting out of bed. The liability to cardiac complications continues for five or six weeks after the exudate has disappeared, and is more likely to be present in those cases in which paralytic symptoms have shown themselves.

Of the remaining complications, only **otitis media**, **cervical adenitis**, and **broncho-pneumonia** are at all frequent. Much less frequent are **lobar pneumonia**, **empyema**, **cardiac thrombosis** (in cardiac dilatation), **nephritis** (early in severe toxic cases), **purpura** (during early convalescence), **hemiplegia** (due to embolism or thrombosis), and **secondary rashes**, which appear one to three weeks from the beginning of the illness, and usually take the form of **urticaria** or a blotchy macular and papular **erythema** most profuse on the limbs.

Diagnosis.—An inflammation of a mucous surface or of the skin, accompanied by the formation of a definite false membrane, is,

in nearly every case, due to the action of the diphtheria bacillus.

Faucial diphtheria.—The diseases most likely to be mistaken for diphtheria are—

1. *Simple tonsillitis.*—If there is any exudate with this condition it consists solely of mucus, loose or inspissated. There is usually considerable local pain, especially on swallowing. The attack may be ushered in by a slight shivering fit.

2. *Catarrhal inflammation of the fauces.*—The inflammation in this condition is extensive but not severe. There is rarely much swelling. There is usually but little definite exudate. The local pain and the constitutional symptoms are slight.

3. *Quinsy.*—The inflammation is one-sided, the swelling often extreme, and the pain severe. There are usually pronounced febrile symptoms, and the patient may be delirious. Thick inspissated mucus may be observed, presenting the appearance of false membrane. Suppuration usually results, an event which is rare in diphtheria.

4. *Simple ulceration of the tonsil.*—This usually follows tonsillitis. The presence of ulceration, unless it be very superficial, negatives diphtheria.

5. *Vincent's angina* is a form of ulcerative tonsillitis which may simulate diphtheria very closely. A membranous-looking exudation is seen upon the tonsil, which may or may not be swollen; this exudate is usually found to lie upon an ulcerated surface, and consists partly of the debris of inflammation, partly of slough. The tonsillar ulcer may be deep and has a clean-cut edge. The constitutional symptoms are slight and the disease is a mild one, except in rare cases in which the inflammation and ulceration spread to other parts of the fauces, the pharynx, and larynx. The exudate has a peculiar and offensive odour. Stained smears of the exudate show microscopically the spirilla and fusiform bacilli which are stated to be the exciting cause of the disease.

6. *Septic inflammation of the fauces.*—There is severe inflammation, often with extreme swelling and the formation of thick, but not membranous, exudate. Sloughing may take place and deep ulceration result. There is usually prolonged fever, and the constitutional symptoms are severe. In one form of this inflammation the lesion consists of very painful inflammatory œdema. The mucous membrane is dusky-red. Occasionally there is also distinct

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erysipelas of the skin of the face or neck. The constitutional symptoms are of sudden onset and may rapidly assume a most serious aspect. The larynx may be invaded.

7. *Scarlet fever*.—The variety most often mistaken for diphtheria is that in which the faucial lesion is marked, but the rash slight and transient or entirely wanting. The inflammation is extensive and thick, yellow exudate is often present. The temperature continues to be raised and the patient may be delirious. The faucial lesion does not yield readily to treatment.

8. *Ulcerative stomatitis*.—The ulcers often present a sloughing surface which at first sight looks like false membrane, but if they be scraped their true character is at once revealed. In severe cases a most offensive odour is emitted which is quite different from that of diphtheria. It should be borne in mind that ulcerative stomatitis affects the tongue, lips, gums, and buccal mucous membrane, which are parts very seldom invaded by diphtheria. The constitutional symptoms may be severe.

9. The wash-leather base of a late *syphilitic ulcer* is not infrequently mistaken for false membrane and therefore for diphtheria, but the presence of ulceration rules out that disease. Other signs of syphilis may be found on examination.

Laryngeal diphtheria.—Almost any disease in which the symptoms of croup are present may suggest diphtheria; but the most frequently mistaken are simple laryngitis, measles, and retropharyngeal abscess.

Simple laryngitis can be diagnosed with certainty only by bacteriological examination. *Measles* often begins with laryngitis, but the buccal mucous membrane shows Koplik's spots. Usually, too, the patient coughs and sneezes and the conjunctivæ are injected. *Retropharyngeal abscess* is frequently accompanied by signs of obstruction of the larynx; but inspection and digital search of the fauces will reveal the swelling, which is usually on one side. Errors of diagnosis in cases of croup can generally be avoided by systematic examination. The fauces of the patient (usually a child) suffering from croup should be inspected in a good light; if any exudate can be seen, even though it be limited and not distinctly membranous, the case may be regarded as one of diphtheria. If no exudate be seen the finger should be passed over the pharynx to the larynx, and search made for any swelling, such as a retropharyngeal abscess.

A *foreign body* may also be detected in this way. If this examination reveal nothing the mouth should be examined for Koplik's spots, and if these be absent a bacteriological examination should be made. In adults and older children the larynx should be inspected with the laryngoscope.

Vulval diphtheria.—The only disease likely to be mistaken for vulval diphtheria is *erysipelas*; but in this there is no false membrane, and the inflamed skin presents a definite margin which is absent in diphtheria.

Apart from the presence of false membrane, the other forms of diphtheria can hardly be diagnosed with certainty except by a bacteriological examination; but diagnosis may be assisted by collateral evidence, such as the presence of an undoubted case of the disease in the same family, house, or school.

The Schick test of natural immunity to diphtheria is described under **SEROLOGICAL DIAGNOSIS**.

Prognosis.—The most important factors in prognosis are the variety of the disease (i.e. whether it be faucial, laryngeal, etc.), the severity of the attack, and the patient's age.

Of the common varieties, the most fatal is the *laryngeal*. Statistics based upon upwards of 56,000 cases of diphtheria of all forms treated in the hospitals of the Metropolitan Asylums Board during a consecutive series of years show that the fatality of cases in which the larynx was not affected was 8·8 per cent., while that of those in which the larynx was involved was 18·9. Though in some of the latter the fauces were also invaded, and death was due to toxæmia and not to interference with respiration, yet it can truly be stated that if the larynx becomes attacked, the chances of death are doubled.

The prognosis of laryngeal cases in which the fauces are not affected is more favourable than that of those in which they are, because in the latter class toxæmia is added to the respiratory embarrassment. Of cases submitted to operation (intubation or tracheotomy), those do best in which the patient coughs up a moderate amount of mucus; those are unfavourable in which the mucus is very scanty or excessive.

In *faucial* diphtheria death is due to the effects of toxæmia. These may appear early in the disease, or late, in which case they are shown by paralysis or cardiac complications. The more extensive and adherent the local exudate and the longer it persists, the worse

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the prognosis, because the greater is the opportunity for the absorption of large amounts of toxin. The most important signs of pronounced toxæmia are albuminuria, scantiness of urine, frequent vomiting, and hæmorrhages into or beneath the skin and from mucous membranes. Apart from vomiting, the patient's skin may become very pallid, and the pulse be irregular, infrequent and feeble. Any of these signs is of serious import, and the more pronounced they are the greater the gravity of the case. Hepatic enlargement, a blotchy erythema occurring within the first few days, and convulsions are also grave indications, but they are not common. The more profound the toxæmia, the more likely is the patient, should he survive the acute stage, to be attacked by paralysis.

When the disease is limited to the *nasal passages* the prognosis is good, and this form is rarely fatal. In conjunctival diphtheria the sight may be impaired in consequence of damage to the eyeball, but as regards life the prognosis is good. Vulval diphtheria is not infrequently accompanied by pronounced toxic symptoms, and the prognosis varies according to their severity.

Diphtheritic gangrenous dermatitis is very fatal; fortunately it is rare.

The *age* of the patient has a most important bearing on prognosis. In general terms it may be stated that up to 25 years, the younger the patient the more likely is a fatal termination to occur or serious complications to arise. The fatality is especially high in children under 2 years. It rises again slightly in persons over 25. Laryngeal diphtheria is seldom met with over the age of 7, but when it occurs in adults it is of very grave prognosis. The probable reason for this is that the symptoms of invasion of the larynx are not pronounced in the adult; serious dyspnoea seldom shows itself, till the smaller bronchi are invaded, when it is too late to adopt methods, however energetic, of arresting the disease.

Of the cases of paralysis, about 13 per cent. are fatal; the remainder recover completely. I have never met with a case in which permanent paralysis occurred.

The most serious cases are those in which the paralysis sets in early and rapidly becomes generalized. If the respiratory muscles, and especially if the diaphragm, are involved, the outlook becomes much worse. Repeated vomiting is also very dangerous, as, too, are cardiac complications, which are prone to

persist in a minor degree for weeks or months.

Lastly, attention must be drawn to the influence of the specific treatment by antitoxic serum. There is now a vast body of evidence to prove that the fatality of cases treated with serum is very much less than that of those which are not. The earlier the treatment is commenced, the much more likely is the patient to recover, and to recover quickly and without complications.

Treatment.—The cardinal principles in the treatment may be summed up in a few words: The use of the specific serum and the enjoyment of absolute rest.

Serum treatment.—The specific serum is an antitoxic serum, that is to say, it acts, so far as is known, by neutralizing in some way the diphtheria toxin which is free in the circulation. There is also clinical evidence to show that it limits the spread of membrane; but there are no reasons for believing that it has any effect on the diphtheria bacilli themselves. It is usually given in doses of multiples of 1,000 units. For a mild case treated on the first day, 2,000–4,000 units will suffice. For more severe cases 8,000–16,000 units will be required; while for the most severe, 16,000–24,000 units are necessary. The whole amount should be given at one time. If after two or three days the exudate shows signs of extending or the toxic symptoms do not abate, the dose may be repeated.

The serum is usually supplied in liquid form. It is not advisable to keep it for longer than two or three months. In circumstances that make it necessary to keep it for a longer period, it is best kept in the form of the dried residue, at a temperature of 5° C., and in a dark room. It can then be preserved with but slight loss of antitoxic value for about five years. For use it should be dissolved in sterilized physiological saline solution.

Serum may be administered intravenously, subcutaneously, or intramuscularly. When given *intravenously* the antitoxin is brought at once into contact with the toxin in the blood, and if it could be easily carried out there can be no doubt that this method would be the most efficacious. But there are serious practical difficulties in the way; and one of the other two methods is usually employed.

The *intramuscular* is slightly the better, for there is evidence to show that the antitoxic portion of the serum is more quickly absorbed. The full effects of the antitoxin are not to

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be expected till after two or three days. The site usually selected is the outer side of either thigh. The skin is painted with iodine solution, and the needle of the syringe is thrust deeply into the muscle. Roux's syringe is the most convenient; but any syringe which will hold 20 c.c. and can be sterilized, will answer the purpose. Strict antiseptic precautions must be adopted. It is well to seal the minute hole made by the needle with collodion.

The beneficial effects of the serum are shown by the reduction of the temperature to normal, the firmer pulse, the limitation of the spread of the false membrane, and the improvement in the appearance of the patient. In nasal cases the discharge ceases.

In some instances effects are produced which are due to the serum and not to the antitoxic principle it contains. In about a third of the cases a rash comes out 7-16 days after the injection; it usually takes the form of urticaria or a hyperæmic erythema of the blotchy or the marginate variety, and is not infrequently accompanied by febrile symptoms. In about 5 per cent. of the cases there is slight multiple arthritis, of which the chief and sometimes the sole symptom is pain in the joints. Occasionally the fauces are slightly inflamed and some of the lymphatic glands, especially those of the neck, are enlarged. Very rarely are the symptoms of this "serum sickness" severe; at the most they are unpleasant; usually they are trivial. In rare cases, symptoms of anaphylactic shock appear; in such they usually show themselves very soon after the injection of the serum. The patients are, apparently, peculiarly susceptible, and there are reasons for believing that asthmatics are more susceptible than others. Persons who have been injected with a like serum more than ten days previously are also prone to exhibit anaphylactic symptoms, which, however, are usually not severe.

Rest is almost as important as the specific serum. The effects of toxæmia may be latent and may be evinced by moderate and even slight exertion. When toxic symptoms have been present or the false membrane has been copious, the patient should be confined to bed for at least three weeks from the disappearance of the exudate. For 10-14 days he should be kept in a recumbent position. When he is allowed to sit up in bed, the effect should be noted, and should there be any acceleration or irregularity of the action of the heart, the recumbent position must be resumed. The

same precaution must be observed when the patient is allowed to leave his bed. The process of leading him on to the full exercise of his muscular powers must be very gradual.

The symptoms of profound toxæmia—frequent vomiting, cardiac irregularity and infrequency, suppression of urine, and hæmorrhages—are, unfortunately, hardly at all amenable to treatment. For the first, full doses of tincture of belladonna (20-30 min. every four hours), should be given combined with the rectal injection of 20-30 gr. of potassium bromide. I have tried many remedies for the other symptoms just mentioned, but have not succeeded in finding one that has been successful. In the cardiac cases stimulants have been most disappointing. Some authorities strongly advocate adrenalin chloride, subcutaneously or by the mouth.

When symptoms of invasion of the larynx arise the patient should be placed in a tent into which steam is allowed to enter; in a small room the tent is not necessary. The question of surgical interference often arises. There are two methods of relieving the distress due to the obstruction of the larynx, **intubation** and **tracheotomy**, the indications for which are marked restlessness and considerable recession of the chest-walls or cyanosis. I prefer intubation. Unfortunately, however, it is a method of treatment not available in private practice; not only does it require special experience on the part of the medical attendant and the nurse, but it demands that the medical attendant should be within a few minutes' call of the patient, who may cough out the tube and become very quickly urgently dyspnoic. Nor is intubation advisable for patients who are at their last gasp when surgical aid is sought.

The patient should be placed on milk or farinaceous foods made with milk for the first few days, or for so long as acute toxic symptoms are present. Afterwards he may quickly return to his ordinary diet.

Patients who are convalescent from severe diphtheria are frequently anæmic and are benefited by iron. In children the compound syrup of the phosphate is most useful.

Since the introduction of antitoxin, **local treatment** has lost its importance, and, especially in the case of small children, reliance is placed almost solely upon the specific treatment. One of the reasons for this is that children resist local treatment, and their struggles lead to exhaustion and are therefore inimical to recovery. Nevertheless, in older children and

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in adults an attempt may be made to remove the false membrane or other local exudate by cautiously peeling it off with forceps or by endeavouring to dissolve it with solutions. The one which has been in use for many years at the Eastern Hospital has the following formula :—

R̄ Sod. bihorat. ʒi .
Sod. bicarb. ʒi.
Pot. chlorat. ʒss.
Sod. chlorid. ʒss.
Tr. lavand. co., ʒi.
Aq. ad Oi.

This solution may be used either as a spray or a douche, or for swabbing. A saturated solution of boric acid may be employed in the same way. Swabbing is not applicable when the exudate has passed from the tonsils to neighbouring structures. When the exudate is localized to the tonsils, swabbing with perchloride of mercury (1 in 2,000) is most useful.

Complete rest is the most essential part of the treatment of *diphtheritic paralysis*. If the patient has been allowed up before the signs of paralysis have appeared, he should be ordered back to bed again, and he should not be allowed to get up until the paralysis has disappeared, and then only with caution, as has been described above for toxic cases. If the patient coughs or splutters when he drinks, nasal feeding should be employed at regular intervals of four hours. The foot of the bed should be raised considerably, so that the patient's head is much lower than his trunk, in order to prevent saliva or any vomited matter from passing into the trachea and bronchi. When the respiratory muscles are paralysed and there is cyanosis, the frequent inhalation of oxygen is beneficial. When there are signs that the patient is beginning to mend, the muscles of the limbs should be massaged. During convalescence the diet should be generous, and strychnine and iron should be given.

Diphtheria of the *vulva* is best treated by the frequent application of hot boric fomentations, and of the *eye* by frequent syringing with boric lotion. The specific treatment should also be employed by the usual method.

Patients suffering from diphtheria should be isolated either in hospital or in a separate room. In respect of the length of the period of isolation, I am in the habit of detaining patients in hospital for three weeks after the disappearance of the local exudation, provided that their condition is otherwise such

DISINFECTION

as will permit of their going out. Some authorities insist that there shall be at least two consecutive negative cultures before a patient is allowed to leave, but the experience of the large fever hospitals of the metropolis goes to show that, so far as the occurrence of fresh cases in the home is concerned, there is no difference between those hospitals in which negative cultures are insisted upon before discharge and those in which they are not. A person may still harbour the diphtheria bacilli in his fauces or nasal passages several weeks or even months after he has recovered from the effect of diphtheria i.e. may become a "carrier." For the treatment of such a case, see CARRIERS OF INFECTION.

E. W. GOODALL

DIPHTHERITIC NEURITIS (see DIPHTHERIA, p. 366).

DIPLEGIA, CEREBRAL (see CEREBRAL DIPLEGIA).

DIPLOPIA (see OPHTHALMOPLEGIA).

DIPSOMANIA (see ALCOHOLISM).

DIRT EATING (see PICA).

DISINFECTION.—This term denotes the destruction of the micro-organism or unknown virus which is the cause of infection. It may be effected—(1) By natural means, such as sunlight, air, heat, and cold, and association with other micro-organisms. (2) By artificial means, such as dry or moist heat, and chemical disinfectants. (3) By a combination of (1) and (2).

Disinfection of the sick-room and its contents.—Beyond ensuring strict cleanliness and adequate ventilation, any attempt to disinfect the room when it is occupied by a patient would be idle. In diseases where the infection is conveyed by discharges from the mouth and nose, such as diphtheria, scarlet fever, measles, whooping-cough, and cerebro-spinal meningitis, special care should be taken to prevent contamination of objects in the room, and all lint, wool, etc., used for absorbing discharges should promptly be burnt. In enteric fever, in which the infection is conveyed by the faeces and urine, these should be well mixed with a liquid disinfectant (e.g. lysol, 1 in 20) and allowed to stand for an hour before being thrown down the closet. Soiled linen, before being boiled or treated with a liquid disinfectant should be immersed in cold water

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to remove grosser impurities and to prevent the stains being permanently fixed. Some form of fly-catcher, especially in enteric fever, should be kept in the sick-room.

In private houses a sheet moistened with a disinfectant is usually hung on a screen round the patient or over the door, for by such means a certain amount of infection is neutralized.

The thoroughness of the terminal disinfection of the room should depend on the nature of the disease and the stage at which the patient left it. More vigorous measures are required if the patient has died in the acute stage than when he has been discharged at the termination of convalescence.

In the case of whooping-cough, measles, and rubella nothing more is needed than an ordinary cleansing of the room and leaving the doors and windows wide open for a few hours.

When a more thorough disinfection is required, as after scarlet fever and smallpox, the room should be made airtight as far as possible and a gaseous disinfectant, such as formaldehyde or sulphurous acid, should be used. Formaldehyde, though very irritating to the mucous membranes, has the merit of being non-poisonous and of not discolouring fabrics or metals except iron or steel; on the other hand, it has no action on vermin, and sulphurous acid should therefore be preferred in the case of diseases known to be conveyed by insects, such as plague, typhus, and relapsing fever. After the fumigation has been completed the doors and windows should be left wide open for at least twenty-four hours.

When fumigation is impracticable, the walls and floors should be scrubbed with a strong disinfectant solution, such as 1-in-2,000 perchloride or 5 per cent. carbolic acid, and the ceiling whitewashed.

Gaseous disinfectants have only a superficial action, and all articles in the room, such as bedding, carpets, rugs, and books will have to be exposed to the penetrating action of steam in a special apparatus. Certain articles like leather or furs, which would be injured by steam, must be treated by hot air. Crockery may be disinfected by being boiled or by the use of a disinfectant solution.

Disinfection of the patient.—This is a much more difficult problem than disinfection of inanimate objects (*see* CARRIERS OF INFECTION). Some authorities indeed—e.g. Chapin, of Providence, U.S.A.—convinced of the supreme importance of the human carrier, have

DISSEMINATED SCLEROSIS

abandoned as useless the terminal disinfection of the room after scarlet fever and diphtheria.

During the course of the illness the nose, mouth, and ears should receive special attention, as they so frequently form the principal sites of infection. Daily baths should be given, but in my opinion no useful purpose can be served by the application of a disinfectant such as eucalyptus oil to the skin.

In the case of scarlet fever and smallpox the patient spends the last night in hospital in a special discharge block, where he is given a hot bath and has his hair shampooed, leaving the hospital on the following morning. Diphtheria patients are often treated in the same way.

In diseases in which the persistence of infection can be ascertained by bacteriological examination, vaccine treatment and local measures have been tried with a varying degree of success, e.g. spraying the throat with formaldehyde or lactic acid, sucking of formamint lozenges or pastilles of bactericidal serum, or the use of a staphylococcus spray.

In every case artificial methods should be supplemented by making the patient remain in the open air and sunlight as much as possible.

Attention should also be paid to his general condition, improvement of which will increase the bactericidal powers of the organism.

J. D. ROLLESTON.

DISORDERED ACTION OF THE HEART (*see* HEART, IRRITABLE).

DISSEMINATED SCLEROSIS (*Insular Sclerosis*).—A common non-systemic disease of the central nervous system, of obscure but probably infective origin, characterized by the multiplicity and variability of its clinical manifestations and by remarkable remissions in its course.

Etiology.—Till recently nothing certain was known about the origin of the disease, but analogical evidence suggested the presence of a chronic infection which manifests itself by repeated attacks on the central nervous system at irregular intervals over a number of years. There is no justification for connecting the disease with syphilis. During the last few years it has been found that the subcutaneous and intrathecal injection of cerebro-spinal fluid and emulsions of the spinal cord from acute cases may produce paralysis in animals, with lesions in many respects similar to those which characterize the disease in man. Kühn and

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Steiner have discovered a spirochæte in the nervous system, blood and liver of the injected animals. A history of influenza or typhoid fever preceding the appearance of the first symptoms is not uncommon, and the writer has seen them develop during an attack of whooping-cough. Trauma does not appear to play any part in its production.

The onset usually occurs between 15 and 50 years of age, and more commonly between 20 and 35. Both sexes are affected about equally, and very rarely two members of the same family may suffer. There is no evidence to show that the disease can be transmitted from one generation to another.

Pathology.—Scattered quite irregularly throughout the brain and spinal cord are found islets or plaques of so-called sclerosis. The more recent of these show signs of inflammation; the older ones may be termed scars. They vary much in size and shape, and their contour does not appear to be determined by vascular supply or by other anatomical features. White and grey matter may be equally involved, and a patch may include both within its area.

To the naked eye a plaque may be distinguished by its semi-translucent greyish-pink appearance, and to touch by its consistency, the latter being firmer or softer than that of surrounding parts.

Under the microscope a diseased area presents features which are quite unlike those of any other morbid process of the nervous system. Nerve-cells, with but few exceptions, escape injury. Nerve-fibres are remarkable for the disappearance of their myelin sheaths and for the comparative integrity of their axis-cylinders. It is true that in long-standing cases there may be a certain amount of destruction or distortion of the axis-cylinders, with secondary degeneration outside the plaques, but, speaking generally, the axis-cylinders are preserved and secondary systemic degeneration is distinctly scanty. Other important features of the sclerotic islet are a compensatory overgrowth of neuroglia, replacing the lost myelin, and the presence of slight thickening and hyalin degeneration of the vessel walls. In the lymphatic spaces of the adventitia may sometimes be found small numbers of lymphocytes as well as migratory cells containing the products of myelin destruction.

The preservation of axis-cylinders just described is worthy of special notice, because it serves to explain the remarkable and rapid

recovery of function which frequently follows the profound effects of fresh patches of disease in the clinical history of most cases. The effect of a morbid patch is to abolish temporarily the function of the neuraxones passing through its territory, but not necessarily to destroy their structure. Areas of sclerosis are not absolutely confined to the central nervous system. As might be expected, they are frequently found in the optic nerves and very rarely in the cranial and spinal roots.

Symptomatology.—A category of all the symptoms and physical signs which have been observed in cases of disseminated sclerosis would not only be a lengthy compilation, but would afford a very poor description of the disease as it is commonly met with. Before attempting, therefore, to deal with separate symptoms, let us draw a sketch representing the most familiar aspects of these cases as they confront us in consulting practice. A young man of 25 seeks advice on account of some difficulty with the right leg, which begins to drag after walking half a mile. He thinks "he must have strained it," but does not remember doing so. "I noticed it first one day after playing tennis." "Have I ever had anything of the kind before? Yes, about two years ago, but the doctor said it was rheumatism; and after rubbing it with some liniment for about a fortnight it got quite well and never bothered me again." "Have I ever had anything wrong with my eyes? Never since I was 19, when the sight of my left eye went all wrong for about a month; but they said it was a chill, and it was quite well after wearing some glasses for a bit."

These few words of conversation in the course of taking the patient's history are so characteristic of the disease that they almost suffice for a diagnosis.

On examination it is found that the right leg is stiff and displays some weakness in dorsiflexion of the ankle and flexion of the knee. The knee-jerk is exaggerated; ankle-clonus and an extensor plantar response can be evoked. The left leg shows similar signs but in much slighter degree, and is regarded as normal by the patient.

An attempt to elicit abdominal reflexes meets with no success. The arms and grasps are quite strong, but when the patient is asked to touch the tip of his nose with the tip of his first finger, the eyes being closed, there is noticed just at the completion of this act a slight "terminal tremor" and perhaps a trifling in-

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accuracy on the part of the finger in finding the nose.

Examination of the cranial nerves may show nothing except some temporal pallor of the left optic disc and perhaps a suggestion of nystagmus.

When the patient walks it is noticed that, in addition to the tendency for the toes of the right foot to catch the ground, the gait is conducted on a wider base than normal, and that any attempt to toe and heel a line is attended with disaster to his equilibrium.

Probably 60 or 70 per cent. of cases of disseminated sclerosis when first seen by the physician conform more or less closely to this picture. He advises some treatment. The patient improves and perhaps returns to active life. A year or two later he turns up with a complaint that his left hand is useless, and that the left leg now is as bad as the right one used to be. It is found that the left arm and hand are strong but quite lacking in sense of position and stereognosis, while both legs are more spastic than they were. In answer to a question he says that his bladder sometimes "hurries him a bit," and that if he has waited there may be delay in commencing micturition. The control, too, of the bowel after an aperient may not be all that could be desired.

And so the story may go on—more or less sudden relapses, with involvement of fresh territory, separated by periods of remission and often of remarkable recovery. But, as time passes, the amount of recovery is lessened and the permanent disability perhaps slowly but surely increases. The relapses are usually attributed—how correctly it is difficult to say—to a stress of work, to a chill, to a shock, or to any of those incidents of life which loom so large in the pathological vision of the public.

Not infrequently the onset of new or the exacerbation of old symptoms is associated with a feeling of malaise and a rise of temperature. Not unnaturally this is regarded as influenza, although it is probably a part of the disease and the natural accompaniment of a fresh crop of inflammatory patches in the nervous system.

Should the first areas of disease be situated in the brain, and especially in the pontomedullary region, there are likely to be severe attacks of headache and vomiting, which, with a slight papillitis, may suggest the diagnosis of an intracranial growth.

There is another type of case, not nearly so

common as that first described, in which a well-marked nystagmus, a coarse intention-tremor of the limbs, trunk, and head, and a characteristic staccato articulation form the prominent features. Such cases attract more attention and less easily escape a proper diagnosis, but they are not nearly so frequently met with as those in which a spastic paraplegia is the only striking feature.

Since early diagnosis is an ideal at which we should all aim, attention may profitably be directed to some of the first symptoms of the disease. The tendency to drag one leg, transient impairment of vision in one eye, astereognosis of one hand, and an attack of headache and vomiting with slight papillitis, have all been mentioned, and may each be the first indication of disseminated sclerosis. The disease may, on the other hand, be ushered in by an attack of partial hemiplegia or of hemianæsthesia, which is often regarded as hysterical. In several instances the patient has described an attack of numbness or paræsthesia affecting the trunk and lower limbs from the waist downwards on one or both sides of the body. This may be associated with an ataxic gait and with various degrees of sensory impairment. It is important to note, however, that there may be no affection of the motor system and no alteration in the reflexes.

In connexion with sensory phenomena, emphasis may be laid upon the fact that, although paræsthesiæ in the form of numbness and pins and needles, associated with slight blunting of sensibility, are common, profound anæsthesia, analgesia, and thermanæsthesia are rare manifestations, and are only met with in the last stages of the disease.

Ocular disturbances.—Diplopia with some degree of ocular palsy and strabismus, generally due to weakness of one external rectus, is a common defect and may be associated with vertigo. Nystagmus, either spontaneous or only elicited on attempts to sustain movements of the eye in one or more directions, is frequently met with, and may be vertical, lateral, or rotatory in character. The pupils may be normal in size and reaction, but occasionally they are unequal, and rarely the Argyll-Robertson phenomenon may be present in one eye. Visual impairment is often observed, and may take the form of transient blindness, concentric narrowing of the fields, or scotomata, especially for colours.

Other cranial nerves.—Slight facial weakness on one side is common. A feeling of numbness

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or coldness in the territory of one trigeminal nerve has sometimes been observed. Disorders of articulation, sometimes called "staccato" or "syllabic," are frequently noticed, and are occasionally accompanied by slight trouble in deglutition. Spontaneous rhythmical movements of the tongue, the palate, or even of the jaw—resembling the ocular nystagmus—are rare phenomena, but similar movements of the head, especially of the "nodding" variety, are by no means uncommon.

The mental condition of patients suffering from disseminated sclerosis is very variable, and probably depends upon the extent to which the disease affects the cortical and sub-cortical regions of the brain. The victim's character, interests, and intellectual acuity are often modified or impaired, and it is generally agreed that he tends to become either lethargic and fatuous, or more often perhaps somewhat facile and unappreciative of the seriousness of his malady. In later stages mental deterioration may be accelerated by the occurrence of epileptiform seizures, and it sometimes happens that his testamentary ability is called in question.

Attention has already been directed to the irregular course of the disease, the long intermissions, and the sudden onset of new or the return of old symptoms. An interval as long as fifteen years may elapse between the first and second attacks, and such an interval may be one of perfectly normal health. More commonly the periods of intermission may be measured in terms of a few months or a few years; there is a general tendency for these to become shorter and less pronounced in their favourable features, and the patient becomes more and more spastic and ataxic, his sphincters less and less under the control of his will, until a bedridden existence is all that he can aspire to. Paralysis is rarely associated with any localized muscular atrophy, and the general nutrition and health of the patient often remain quite good until some complication or inter-current disease brings about a fatal termination.

Diagnosis.—This cannot depend on the presence or absence of any particular symptom or physical sign. A careful inquiry into the patient's history generally provides important information in that it reveals the characteristic remissions and relapses. A story of steadily progressive deterioration of nervous function in the form of spastic or ataxic paraplegia is more suggestive of *subacute combined degenera-*

tion of the spinal cord or of some *spinal compression*.

It is a common mistake to regard the improvement and recovery which are frequently observed, after the early patches of sclerosis have produced serious disabilities, as evidence in favour of the condition being functional and not organic. It is most important to avoid this error, and to realize that amelioration in the patient's condition may be looked for with confidence. The disappearance of all organic signs is by no means uncommon and does not in any way throw doubt upon the diagnosis.

Perhaps *cerebro-spinal syphilis* is the disease which may be most frequently and most legitimately confused with disseminated sclerosis. It shares with the latter the tendency to remissions and relapses when the patient is treated with anti-syphilitic remedies only on the development of symptoms, and when no attempt is made to prevent further manifestations of the disease. It has, moreover, the same tendency to develop lesions at different sites; the patient may present at intervals a hemiplegia, a paraplegia, or perhaps an oculomotor palsy. Examination of the cerebro-spinal fluid, which in disseminated sclerosis shows no abnormal features, is generally sufficient to prevent a wrong diagnosis being arrived at. Attention has already been drawn to the occasional occurrence of headache, vomiting, and papillitis in disseminated sclerosis, a combination of symptoms which suggests an *intracranial growth*. The rapidity with which these phenomena clear up serves to exclude the presence of a neoplasm. Speaking generally, the diagnosis of disseminated sclerosis is determined by the characteristic history and by the presence of signs pointing to more than one focal lesion in the central nervous system.

Prognosis.—When there is evidence that a patient has suffered from one or more relapses the ultimate outlook must always be regarded as grave, although a certain degree of temporary recovery may be expected. It is impossible to say whether a patient who has suffered from disseminated sclerosis ever makes a complete and permanent recovery. He would eventually escape from medical observation and no record of his case would be forthcoming. At any rate, we know that an interval of good health as long as fifteen years may pass between one relapse and the next. Although the prognosis is grave in all cases, we are not justified in saying that the disease is incurable

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or that there is no prospect of permanent recovery. In rare instances a fatal termination is reached in the course of a few months or a year or two. The large majority of patients live for at least fifteen or twenty years after the first signs of disease have presented themselves.

Treatment.—Any disease which has a natural tendency to relapses followed by remissions presents serious obstacles to a correct estimation of the value of remedies, and disseminated sclerosis undoubtedly affords as good an example of this difficulty as can be found in the whole range of medicine. There seems, however, to be a general opinion that the administration of arsenic favours recovery from the effects of this disease and has some influence in preventing relapses. The writer subscribes to this opinion, and believes that the more or less continuous administration of arsenic is attended by results superior to those of any other method of treatment. He is in the habit of prescribing three or four minims of Fowler's solution three times daily for two or three weeks in every month, and to continue this line of treatment for years. During the last ten years he has often used intravenous and intramuscular injections of arsenical compounds, but, although results have sometimes been favourable, they have afforded no conclusive proof that these methods are more efficient than taking the drug by the mouth. Mercury is another drug which has been extensively used and is advocated by some physicians either in the form of inunctions or by intramuscular injections.

Symptomatic treatment must be based on re-education. The patient must be made to realize that he must be the chief agent in recovering power or co-ordination. On his own efforts more than on anything else depends the restoration of utility in his limbs. Carefully regulated exercises should be prescribed for daily use, care being taken that serious fatigue is not induced. When ataxia is a prominent feature of his condition the use of Frenkel's exercises may be advised. Massage and passive movements are only of use when great rigidity has to be overcome, and even then great care is necessary to avoid overstimulation of reflex action by the manipulations. Electrical therapy cannot be recommended except in the rare instances in which muscular atrophy is present.

Defects of sphincter control are sometimes influenced favourably by the administration of

DIVERTICULITIS

tincture of belladonna in doses of 3-5 min. two or three times a day. The treatment of the later stages of this disease is largely a matter of careful nursing and attention to the general health of the patient.

E. FARQUHAR BUZZARD.

DISTICHIASIS (*see* EYELIDS, AFFECTIONS OF).

DIVER'S PALSY (*see* CAISSON DISEASE).

DIVERTICULITIS (*syn.* Perisigmoiditis; Pericolitis Sinistra).—Although the term diverticulitis is sometimes applied to inflammation of Meckel's diverticulum, it usually denotes an inflammation of sacculi of the colon, especially of its pelvic part.

Etiology.—The sacculi are multiple and may occur in any part of the colon, but are commonest in the sigmoid flexure. There is some difference of opinion as to whether they are congenital in origin, but symptoms appear only in people past middle life. The sacculi consist of protrusions of the mucous membrane through the muscular layer, where it is perforated by the blood-vessels, and subsequently along their sheaths, usually into appendices epiploicæ. A deficiency of the non-striated muscle is probably contributory to the further weakening of these comparatively undefended areas. Increased tension within the bowel, as by constipation and chronic obstruction, probably also plays a part in the etiology. The sacculi are generally small and harmless, but diverticulitis may result from faecal concretions and from constriction of their necks, with subsequent infection.

Symptoms and course.—With inflammation of one or more diverticula there arise pain, rigidity, tenderness and swelling in the left lower quadrant of the abdomen, the condition closely resembling appendicitis, though situated on the other side. Such attacks may recur on several occasions, but usually subside satisfactorily. On the other hand, an abscess may be formed or a faecal fistula, which may communicate with the bladder or vagina, or, more rarely, may open externally. Other effects of perforation may be general or local peritonitis. If a continuous chronic inflammation or repeated attacks cause hyperplastic and fibrotic changes, a considerable tumour may be produced, which is hard, irregular, often mobile, and sometimes adherent to the surrounding structures, and not distinguishable, in some cases, from carcinoma, even at an operation.

DRUG ERUPTIONS

Obstruction may occur in these circumstances. Carcinoma itself would appear to be an occasional sequel.

Diagnosis.—With inflammatory signs resembling those of appendicitis, but limited to the left lower quadrant of the abdomen, diverticulitis should be borne in mind, especially in the case of old people who give a history of previous attacks. In women, pelvic inflammation may be closely simulated. To distinguish the chronic proliferative form from carcinoma of the sigmoid the following points may help: (1) A history of previous attacks; (2) earlier incidence of the pain; (3) obesity, or at least better maintenance of nutrition despite a long history; (4) absence of blood in the stools; (5) detection by cystoscopic examination of a vesical fistula which appears to be inflammatory; (6) no evidence of malignant growth by sigmoidoscopy. X-ray examination may afford a clue to the right diagnosis by failing to show the usual shadows of malignancy. In the non-obstructive cases a barium meal, or enemata containing barium, may demonstrate by this means that faecal material is retained in diverticula after the remainder has been passed.

Treatment.—For the simple inflammatory form, rest in bed and careful dieting, combined with hot applications to the abdomen, saline purgation, and gentle lavage by enemata of warm water, usually suffice. Unless the attacks recur frequently and are very severe, surgical treatment is unnecessary. When this is the case, or when fistulae or obstruction are produced, excision of the whole sacculi-bearing part of the colon is the only remedy. In the presence of obstruction, preliminary colostomy or ileo-sigmoidostomy is advisable.

FREDERICK LANGMEAD.

DROPSY (see **CEDEMA**).

DROWNING (see **ASPHYXIA**).

DRUG ERUPTIONS.—Drug eruptions are of two classes: (1) those of external origin, (2) those of internal origin.

1. DERMATITIS DUE TO DRUGS APPLIED TO THE SKIN

The epidermis is the protective layer of the skin, and an intact epidermis has greater resistance to irritants than one which is damaged by injury. Maceration of the epidermis produced by the prolonged application of poultices, plasters, and oily liniments renders the skin liable to infection by pyogenic

organisms. The normal epidermis, however, is unable to withstand certain forms of irritation, and there are numerous drugs locally applied which cause inflammation of the skin. In some cases individual susceptibility is a striking feature. The commonest drugs responsible for cutaneous irritation are the following:—

Boric Acid.—This is the least irritant of antiseptics, but even boric acid is not tolerated by some persons and causes an erythematous eruption.

Carbolic Acid, even in a weak solution, causes an erythema and sometimes vesication with cedema. In a strong solution it acts as a caustic, producing a white eschar.

Formic Aldehyde makes the epidermis tough and dry, and causes desquamation. A dermatitis of eczematous type may also occur, and occasionally necrotic areas have been observed.

Iodoform applied to wounds in certain susceptible persons produces an erysipeloid eruption in the surrounding parts. The skin is intensely congested and covered with small vesicles, and there is much cedematous swelling. These phenomena are especially seen when the face, scrotum, or vulva is affected. The eruption may spread widely from the site of irritation. The erythema is followed by desquamation, and occasionally suppuration occurs. Symptoms of general intoxication, even with fatal results, are reported.

Aristol, Airol, Europhen, Iodol, and Orthoform may produce a somewhat similar affection in certain subjects.

Mercury and Salts of Mercury cause erythema with itching, followed by minute, closely-set vesicles and pustules. In mercurial inunction care must be taken to avoid the hairy parts, as there is a great tendency to the production of a pustular folliculitis. This is not unusual after the common practice of treating pediculosis pubis by unguentum hydrargyri. The least irritant mercurial salt is the bichloride of mercury and zinc.

Salicylic Acid in some subjects is an irritant, and ointments containing a rather high proportion of this drug (30 gr. to the ounce) may cause intense smarting and burning, with erythema and vesication.

Arnica in the form of a tincture, a popular remedy for bruises, etc., may cause an acute, widely spreading eczematous eruption.

Atropine.—The application of atropine and belladonna may, in the susceptible, cause an acute erythema, with vesication and cedema.

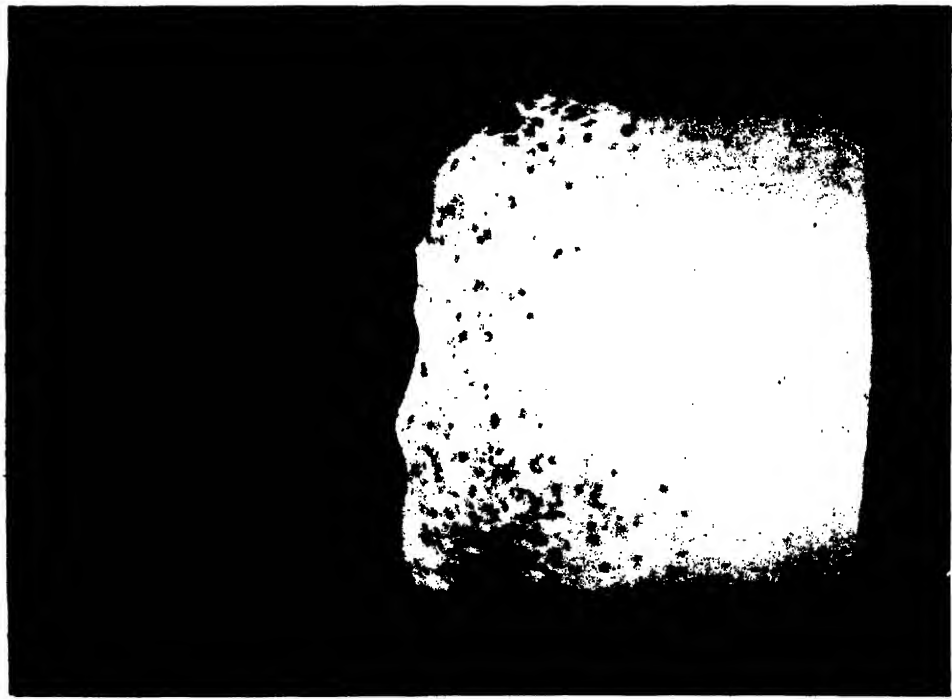


Fig. 1.—Group comedones in child, due to rubbing in camphorated oil.

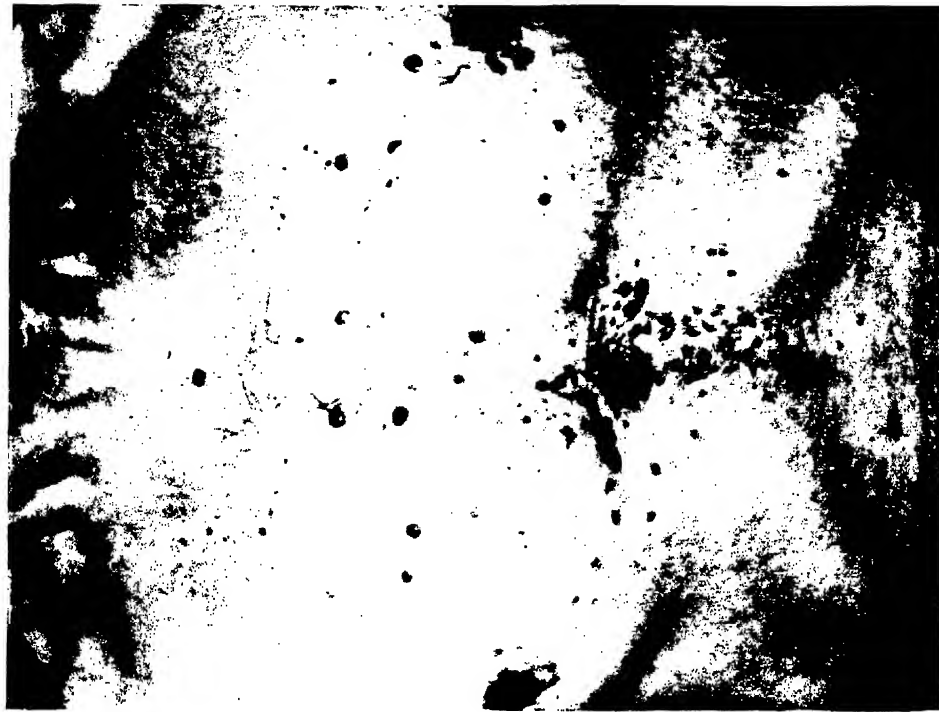


Fig. 2.—Comedones in male aet. 58, due to rubbing in castor oil and mustard.

PLATE 9.—DRUG ERUPTIONS.

DRUG ERUPTIONS

In ophthalmic practice the eruption spreads from the lower lid on to the cheek.

Oil of Cade, often used instead of common tar, may cause a dermatitis characterized by erythema and vesication. Its prolonged employment causes a thickening of the horny layers of the epidermis. In some persons this, like the other tars, produces an eruption of pustules in the hair-follicles.

Cantharides is used as a vesicant, as a hair stimulant, and as a rubefacient. In certain subjects it may evoke an acute vesicular dermatitis.

Chrysarobin, employed in the treatment of psoriasis, tinea, etc., causes an intense erythema of a prune-juice colour, with itching and burning. If the drug is used on the face, there is intense cedema, with conjunctivitis and swelling of the eyelids. The eruption may last for several weeks, and is succeeded by a bronze staining.

Naphthol, if too strong, produces a dermatitis. It must be used with care in young children.

Peroxide of Hydrogen, if too concentrated, causes redness and vesication.

Picric Acid, which is usually considered free from risk, may occasionally cause an eczematous eruption.

Pyrogallic Acid stains the skin a brownish-black colour, and in certain subjects causes an acute dermatitis with great swelling. In strong solution it is an escharotic.

Sulphur dermatitis is very common. It is frequently seen after the treatment of scabies, especially in a zealous patient. The sulphur causes itching, and the patient fearing that his scabies is not cured, continues to rub in the ointment until an eczematous eruption is produced.

Tar eruptions have been described above (see Oil of Cade).

The lesions that may be produced by the rubbing in (a) of camphorated oil and (b) of castor oil and mustard are illustrated in PLATE 9.

It must be remembered that certain antiseptics used in dentifrices may be irritants; a troublesome eruption about the lips may be due to this cause.

Hair dyes.—The commonest in use are peroxide of hydrogen, nitrate of silver, and pyrogallic acid, which may occasionally cause irritation. Paraphenylenediamine, however, is the most dangerous application. In many instances it can be used with impunity for a time, and then an acute, intensely irritant dermatitis follows the application. The fore-

head and eyelids become much congested and cedematous, and the whole face and neck and even the shoulders may be involved. In severe cases the area becomes vesicular and weeping, and crusts form. The inflammation lasts for several days, and is followed by scaling.

The **treatment** of these various forms of dermatitis consists in (1) the removal of the irritant, (2) the application of soothing lotions and emollient powders.

2. DERMATITIS DUE TO THE INTERNAL ADMINISTRATION OF DRUGS

Idiosyncrasy plays an important part in these eruptions. In some cases a small dose is sufficient to cause an outbreak. In others there is accumulation of the drug owing to repeated doses, or to deficient elimination as in chronic renal disease.

The eruption may be an *erythema*, in patches, or a diffuse *roseola*, sometimes associated with congestion of the fauces and of the conjunctivæ. It may spread widely, but usually subsides in a few days with little or no desquamation.

Such an eruption may be caused by antimony, antipyrin, arsenic, benzoic acid, boric acid, bromide, chloral, copaiba, cubebs, digitalis, exalgine, iodides, morphine, quinine.

A less common form is a *scarlatiniform erythema* which may involve the whole surface or be limited to the trunk and flexures. It is followed by desquamation. It occurs after the administration of antipyrin, arsenical compounds such as arsenobenzol, etc. (given intravenously), belladonna, benzoic acid, chloral, ipecacuanha, nux vomica, opium, quinine, sulphonal.

Urticaria may occur after antipyrin, arsenic, bromides, chloral, iodides, morphine, quinine, and santonin.

General exfoliative dermatitis may follow repeated injections of the arsenical compounds used in the treatment of syphilis.

Purpura may occur after the administration of antipyrin, arsenic, belladonna, chloral, copaiba, ergot, iodides, mercury, phosphorus, quinine, and salicylates.

Bullæ are specially seen in bromide and iodide eruptions, and rarely in poisoning by aconite, antimony, antipyrin, arsenic, quinine, salicylic acid.

Pigmentation is caused by arsenic, silver and antipyrin.

Gangrene may be due to poisoning by ergot, antipyrin, arsenic, chloral, iodides.

DRUG ERUPTIONS

Alopecia is produced by salts of thallium.

The **diagnosis** of these eruptions is often attended with difficulty, as one drug may cause several types of dermatosis. The sudden onset of the eruption, the peculiar characters, and the absence of evidence of the usual concomitant symptoms of other conditions, may arouse suspicion, but in all doubtful cases careful inquiry must be made as to what medicines the patient may have been taking—and it must be remembered that many people take drugs which are not ordered by their medical attendants.

The special features of eruptions due to certain commonly used drugs demand particular attention :

Antipyrin rashes.—These are most commonly of the erythematous type, either in patches or diffuse, and occasionally macular. Urticarial eruptions and purpura also occur. Brocq describes as pathognomonic a variety of erythema in round or oval patches, scabbed over the surface, of a dull-red colour, varying in size from a small coin to an area as large as the palm of the hand. The redness passes off in a few days, and is followed by a fine desquamation and a brownish stain, which fades very slowly. Successive attacks follow the re-administration of the drug. In rare instances blebs and gangrene have been reported.

Arsenic may cause erythema, urticaria, purpura, vesico-bullous eruptions, and also herpes zoster. After repeated intravenous injections of arsenobenzol, novarsenobenzol, and similar arsenical compounds, a grave form of general exfoliative dermatitis is not uncommon. The prolonged internal administration of arsenic produces a diffuse or patchy pigmentation, especially of the trunk. In rare cases there is a remarkable hyperkeratosis of the palms and soles, of a yellowish-brown colour, sometimes with warty excrescences which may develop into arsenical cancer.

Bromide eruptions.—The commonest type is an acne, which occurs frequently in epileptics and others taking large doses of bromides. In some cases the eruption consists of pustules covered with a dirty-yellowish crust resembling ecthyma. In young children a remarkable eruption of characteristic type consists of large, well-defined, round, soft swellings which often have a suppurating border. These vegetations develop with great rapidity, and by the confluence of adjoining lesions may cover consider-

able areas. They may occur anywhere, but I have seen them most frequently on the lower limbs and buttocks (Plate 10, Fig. 1), and occasionally on the face.

Copaiba eruptions.—These are either generalized or patchy erythemas, and are attended with intense itching. They are of a bright-red or pink colour and involve the trunk and extremities.

Cubebs and Sandal-wood Oil may cause similar manifestations.

Iodides.—Erythematous and urticarial eruptions of diffuse character and purpura are seen in certain subjects after the administration of even small doses of the drug. In some instances an eruption resembling erythema nodosum may occur. Like the bromides, iodides may produce an acne closely resembling acne vulgaris. The most characteristic iodide eruption is bullous (Plate 10, Fig. 2). The blebs may contain clear serum, but more often the fluid becomes purulent; the central parts form vegetations and are covered with crusts. The lesions develop very rapidly and closely resemble a variety of syphilide. The face, neck, flexures of the limbs and trunk are most often involved. The eruption may last for several weeks, and if the drug be pushed the lesions ulcerate, and may extend to the mucous membranes. There may be grave general symptoms, albuminuria and diarrhoea, with fatal results. The worst cases I have seen have been those in which there was grave cardiac and chronic renal disease.

Mercury in certain subjects, even in small doses, may produce toxic symptoms. The eruption may be an erythema, with occasional vesicles in the flexures. In more severe cases there may be weeping areas, hæmorrhages and pustules. The irritation is intense, and there may be albuminuria, fever, and gastro-intestinal symptoms. In rare cases death has resulted.

Treatment.—The essential point is to stop the medicine which is producing the toxic effects. The bowels should be kept acting freely, and the patient should take plenty of fluid. Intramine is of value in the grave arsenical dermatitis following intravenous injections. Local treatment depends upon the nature of the lesion. The erythematous and urticarial types are relieved by the application of powders and soothing lotions. Bullous lesions should be kept dry with powders. (See also the Treatment section of ECZEMA.)

J. H. SEQUEIRA.



Fig. 1.—Bromide eruption.



Fig. 2.—Iodide eruption.

PLATE 10.—DRUG ERUPTIONS.

DRUG HABIT

DRUG HABIT.—This subject will be considered in relation to the craving for morphia, cocaine, ether, and chloral.

Morphinomania.—This may be defined as an irresistible yearning for morphia. The symptoms produced by habitually smoking opium, or by taking it by the mouth for other than medical purposes, closely resemble those produced by the subcutaneous injections of morphia, and as this latter mode of the abuse of opium or its derivatives is commonest in civilized communities, it will here be particularly described.

Etiology.—Morphinomania has its chief incidence upon the neurotic, degenerate and ill-balanced, who have tried the drug out of curiosity, or have been medically introduced to it for the relief of pain and have unhappily continued its use after the need for it has ceased. The professions having peculiar knowledge, such as those of medicine, pharmacy, and nursing, yield a specially large number of victims.

Symptoms.—For an indefinite period, varying from weeks to years in different subjects, the only results of the habit are harmless. After each dose there is a feeling of physical and mental exhilaration, the affective tone is raised, and the intellectual powers are increased. These gratifying results are, however, purchased at the cost of subsequent depression and weariness, to overcome which the patient again has recourse to the drug. As time goes on, the amount required to produce the desired result has to be increased, while the time elapsing between the doses is gradually decreased. In this way the habit is formed, a state of chronic intoxication is produced, and sooner or later symptoms appear. The patient becomes indifferent to anything save the drug, and will sacrifice everything to procure it. The tone of his character gradually becomes lowered, and he is apt to be guilty of acts of indelicacy, impropriety, and even of crime. His power of attention, his memory, and his will-power become feeble. The unpleasant sequelæ of a dose become continually accentuated and, unless one is speedily followed by another, restlessness, irritability, anxiety, agitation and intense weariness develop. Insomnia is pronounced. The pulse accelerates and is small, and there may be cold sweats and a tendency to syncope. As the dose is increased and the intoxication becomes more profound, the general nutrition is more and more impaired. Anæmia and emaciation occur. There are

breathlessness, feebleness of the action of the heart, and disturbances of the alimentary tract, such as vomiting, diarrhoea, and constipation. Miosis is common. Occasionally there is hyperæsthesia and occasionally anaesthesia of the extremities. The reflexes are diminished. Albuminuria sometimes develops. Abscesses often occur, from the use of dirty needles. Sleep is usually poor, and disturbed by dreams of an unpleasant character. An acute confusional state may arise in this as in other intoxications, but often the confusion is of a mild and chronic variety, with hallucinations and delusions. Depression is frequent. If the habit is persevered in, the mental faculties become feebler until dementia is definite, and eventually death occurs from cachexia, cardiac failure, tuberculosis, or septicæmia from dirty punctures.

Treatment.—The essential treatment of morphinomania consists in the suppression of the drug. This may be effected suddenly or over a longer period. Sudden suppression is a most dangerous procedure, and may result in cardiac failure. It should never be attempted in the case of those over middle-age, or whose hearts are not of the strongest, or who are cachectic or in any serious state of physical health. The sufferings of deprivation are less severe when the withdrawal is extended over a period of weeks, though at the same time they are more prolonged; the danger of this plan is far less than when withdrawal is sudden. The patient having been put to bed, the full dose to which he has been accustomed should be given on the first day and thereafter daily reduced by one-tenth, so that on the eleventh day no morphia is given. The restlessness and mental and physical discomfort resulting from deprivation may be alleviated by the administration of hyoscine, bromide, veronal, amylene hydrate, or paraldehyde. A careful watch should be kept on the heart, and if any sign of failure develops, appropriate remedies should at once be employed. When the drug has been finally withdrawn the patient should be regarded as convalescent from a serious illness, and should gradually be nursed back to health. In some instances suggestion under hypnosis fortifies him against relapses.

The Cocaine, Ether, and Chloral Habits.—The cocaine habit is even more dangerous and more fatal than the morphia habit. The symptoms are very similar. In some cases there is a state of intoxication resembling that

DUPUYTREN'S CONTRACTURE

of alcohol. Hallucinations of sight and hearing are particularly prominent features. Sensations as if the skin were covered by numerous moving insects or small animals are frequent. Diplopia, amblyopia, and dyschromatopsia occasionally occur. Delusions of persecution are frequent, and may lead to assault and homicide. Complications such as agitation, confusion, and cardiac failure occur as in morphia intoxication. The yearning for ether is not so intense as for morphia or cocaine, and the symptoms resemble those of alcoholic intoxication. The symptoms induced by *chloral* chiefly affect the heart and gastro-intestinal system. Mental symptoms, other than gradual enfeeblement, are not common.

The treatment of the cocaine, ether, and *chloral* habits is similar to that for morphinomania.

E. D. MACNAMARA.

DUCHENNE'S PARALYSIS (see MYOPATHY).

DUHRING'S DISEASE (see DERMATITIS HERPETIFORMIS).

DUMBNESS (see DEAF MUTISM).

DUODENAL ULCER (see GASTRIC AND DUODENAL ULCERATION).

DUPUYTREN'S CONTRACTURE. — A slow inflammatory process affecting the palmar fascia and causing a flexion of the fingers at the metacarpo-phalangeal and proximal interphalangeal joints. The finger affected most often is the ring finger, but the little finger is also frequently involved. The contraction is of the longitudinal fibres of the palmar fascia, which come from the insertion of the palmaris longus, and are inserted partly into the fibrous flexor sheaths and partly into the sides of the second and first phalanges. In most cases the metacarpo-phalangeal joint becomes flexed first, and the interphalangeal joint later on. After some time the lateral and anterior ligaments of these two joints shorten up and present an obstacle to complete extension of the finger, even when the shortened bands of palmar fascia have been divided or removed. The flexor tendons escape.

The first sign is an induration at the base of the affected finger and inability to extend it completely. The hardening soon becomes a slightly tender nodule, and the skin adhering to it gets puckered. As the disease progresses the finger becomes more and more flexed until,

finally, its tip comes to rest permanently in contact with the palm.

The **etiology** of the condition is obscure. Apparently pressure on the palm from the use of instruments or tools is not a cause. The deformity rarely occurs before the age of 35, is seen only in men, and the subjects of it are frequently gouty.

Treatment.—In the early stages increase of the deformity may be prevented by the use of a dorsal Adam's splint if this be worn every night. Apart from this, no other treatment short of operation has the slightest influence in checking the progress of the disease. Injections of fibrolysin are useless. Operative treatment is recommended in all but the slightest cases, but a great deal of care and judgment is required to get the best results. Two procedures are recommended: subcutaneous division of the thickened bands, and removal of the contracted fascia by open operation. The *subcutaneous operation* gives excellent results, although the after-treatment requires more care and supervision than that of the open operation. An Adam or similar splint must be worn at night for six months if the results are to remain permanent. Too many, rather than too few, divisions of the fascia should be made, the skin should be separated from the contracted bands by turning the knife on the flat, and if necessary the deformity should be rectified at two or even three sittings. There is only one danger: a clumsy operator may divide one of the digital nerves when he is attacking the prolongations at the sides of the finger; but this accident need not happen. It causes a permanent anæsthesia of a region of the finger at the tip. The *open operation* is completed at one sitting, and no prolonged after-treatment is necessary; but it is liable to drawbacks. After it has been separated the ill-nourished adherent skin may lose its vitality and slough, when the resulting raw area as it cicatrizes reproduces the deformity. Sepsis may produce a like evil result—no very remote possibility in view of the difficulty of disinfecting the creased palm in these cases. When the deformity is very great it is not possible to gain access in order to make the incision, and the subcutaneous operation must perforce be done.

C. A. PANNETT.

DUST DISEASES (see PNEUMONOCOINOSSES).

DWARFISM (see INFANTILISM).

DYING DECLARATIONS

DYING DECLARATIONS.—Deliberate statements of a person who is actually dying, which may be important evidence in cases of death by violence. In order that such declarations may be admitted as evidence the following conditions must be satisfied:—

1. A prisoner must be on trial for the murder or manslaughter of the person who made the declaration.
2. They are only accepted as evidence as to the actual circumstances of death.
3. The declaration may be made orally or in writing; if the former, it must be written down by the person receiving it, and signed or assent otherwise indicated by the dying person after it has been read over to him.
4. At the time the declaration is made the person must be in complete possession of his senses and must believe that he is dying.

These rules will indicate the duty of a medical man, but he should clearly understand the obligation of writing down the actual words used by the dying person. Leading questions should not be put, and, if questions are necessary to make clear any statement, then both question and answer should be recorded. The declaration should be signed in presence of witnesses when possible.

A. ALLISON.

DYSARTHRIA (see SPEECH, DISTURBANCES OF).

DYSCHEZIA (see CONSTIPATION; DIARRHŒA).

DYSENTERY.—A clinical term applied to a group of specific diseases mainly caused by either bacteria or protozoa, and characterized by inflammatory changes in the large bowel and the passage of frequent loose stools containing mucus and often blood.

Two or three centuries ago dysentery caused a high mortality in London and other European towns, but, thanks to improved sanitation, it is now a comparatively rare disease in temperate climates. Serious epidemics have occurred, especially under war conditions and in jails, while outbreaks still appear in asylums because of the filthy habits of the inmates. In tropical and subtropical climates the disease is more sporadic in nature, with a great seasonal increase in the damp hot rainy season. All the earlier descriptions were based solely on clinical experience, for it is only during the

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last few decades that the dysenteries have been differentiated into distinct specific diseases, by far the most important of which are the bacillary and the amœbic forms. As we now know that it is only the latter form which is frequently complicated by abscess of the liver, it is possible by the aid of this test and the recorded descriptions roughly to subdivide the known outbreaks of dysentery into these two classes, as shown by the writer in his book on dysenteries. We thus find that the old epidemics in armies and jails, including those in Indian prisons, as well as more recent asylum epidemics, were of the bacillary type, whilst the sporadic forms of tropical and subtropical climates, with their frequent hepatic complications, were mainly amœbic in nature. During the Great War, dysentery was very prevalent in the Eastern areas, the earlier outbreaks in Gallipoli and Egypt having been largely amœbic in nature, although later the bacillary type prevailed both there and in Greece. In Mesopotamia and East Africa also both types occurred.

The differentiation of the dysenteries began with the recognition of the part played by the amœba in the disease. Lösch, in 1875, first described living intestinal amœbæ as the cause of dysentery, although Lewis and Cunningham appear to have first discovered intestinal amœbæ, probably *Entamoeba coli*, while working at cholera in India in 1870-71. Kartulis, in 1885, attributed dysentery in Egypt to amœbæ, and also first found this organism in the pus of a liver abscess in 1887, while Councilman and Laffleur, in 1891, described the intestinal lesions of amœbic dysentery, which is now known to be a common variety in tropical and subtropical climates. Recently, a few cases have been found to originate in temperate countries, including Great Britain. It was not until 1898 that Shiga established the part played in the etiology of bacillary dysentery by the bacillus which bears his name. In 1900 Kruse independently isolated the dysentery bacillus in Europe, and Flexner found in the Philippine Islands the variety named after him. These two forms are the most frequent and important, but, in addition, there are certain rarer causes of dysenteric symptoms, which must be briefly considered.

The *Balantidium coli* is a large oval parasite, measuring 50-100 microns in diameter, with a peristome at the anterior end and cilia around the rest of the body, an oval nucleus and contractile vacuoles. It multiplies by

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transverse division, but rapidly encysts (Fig. 20) or dies outside the body. It has been found deep in the tissues of the large gut and produces inflammation and ulceration, especially in the sigmoid flexure and rectum. Healthy pigs have been found to harbour it. The form of dysentery which it causes is met with occasionally in the tropics, and especially in the Philippine Islands. Emetine is said to be useless. The prognosis is bad in well established cases, but calomel, salines, arsenic, thymol and enemata of quinine have been recommended in its treatment.

The *Bilharzia hæmatobia* produces dysenteric symptoms when the mucous membrane of the rectum is extensively thickened and ulcerated and contains ova, which may be found in the discharges. Christopherson has shown that bilharzial disease can be cured by a course of full doses of tartar emetic intravenously, totalling 15-25 gr. (See SCHISTOSOMIASIS.)

Dysenteric symptoms have also been attributed to the presence of intestinal worms, which may cause the passage of some mucus, but a microscopical examination of the stools will reveal their ova and lead to appropriate treatment being adopted.

The question remains how far the well-recognized forms of dysentery account for this group of symptoms, and whether any forms remain to be differentiated. Recent research in Indian jails has shown that a considerable number of cases occur in which the organisms neither of amœbic nor of bacillary dysentery can be found, and after allowing for the difficulty of isolating dysentery bacilli in chronic cases, certain of them remain unaccounted for. These are mostly of a mild nature readily yielding to purgative treatment, so it is

highly probable that they are due to a form of simple catarrh. The precise causes of such groups of mild dysentery cases remain to be worked out.

BACILLARY DYSENTERY

An inflammation of the mucous membrane of the large bowel, caused by special bacilli of the typho-coli group, and characterized by frequent stools of mucus and blood, these being generally accompanied by tenesmus.

Etiology. — The dysentery bacillus resembles closely that of typhoid, except that it is free from cilia. It is agglutinated by the blood of dysentery patients except in the early stage of the disease. In addition to that described by Shiga and Kruse, Flexner, Strong and others have obtained varieties which ferment mannite, a property not possessed by the Shiga type. The mannite group is further subdivided by slightly differing fermentation reactions. The bacilli, as a class, grow readily on gelatin, agar, potato, etc., and in milk produce an alkaline reaction following slight early

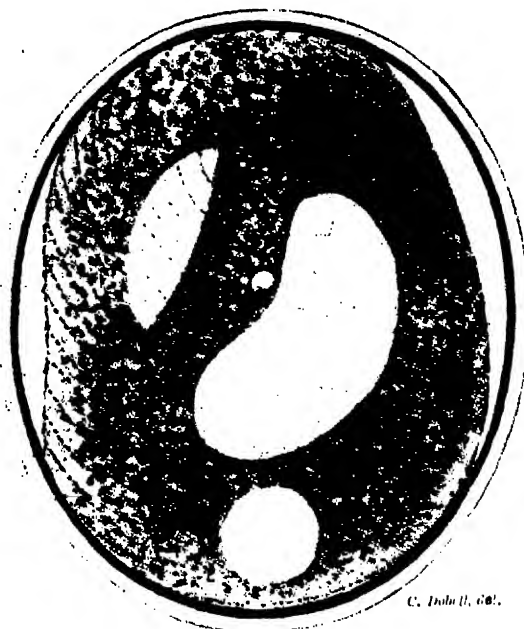


Fig. 20.—Recently-formed cyst of *Balantidium coli*, at stage of development usually seen in freshly-passed feces. $\times 2,000$.

(From Report No. 51 of the Medical Research Council, by permission of H.M. Stationery Office.)

acidity. The Shiga bacillus contains a powerful intracellular toxin. The dead organisms when injected subcutaneously or intraperitoneally into rabbits kill them, lesions in the large bowel resembling those of acute bacillary dysentery being found. By injecting horses with gradually increasing doses of the toxin a powerful curative serum is obtained.

The organism gains access to the body through food and water, and flies act as carriers. It has but slight resisting powers outside the body. Patients with chronic dysentery are probably sources of the infection, so dysenteric stools should be carefully disinfected, prefer-

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ably with a chemical which will repel flies, such as fresh chlorinated lime.

Pathology.—The character and distribution of the lesions in the large bowel in bacillary dysentery vary much with the stage of the disease. In the more acute cases, proving fatal within less than a month, usually the whole or nearly the whole of the large gut is involved, and commonly the lower foot or two of the ileum also. The mucous membrane shows general thickening and congestion, and presents a granular condition due to a fibrinous inflammation of the mucous coat, but there is little involvement of the submucous layer. The most affected parts, especially the summits of the rugæ, then slough away to form irregular depressed ulcers. The process is usually most extensive in the lower half of the large gut, and especially involves the sigmoid flexure and rectum. This explains the frequency of straining and tenesmus in bacillary dysentery. Microscopically the mucous membrane shows a thick deposit of fibrinous material, a small-celled infiltration, destruction of the tube glands, and the presence of numerous bacteria. In chronic cases of one to many months' duration, extensive lesions are generally confined to the lower half or two-thirds of the large gut, the upper part being often fairly healthy. The affected portion shows a generally thickened mucous membrane studded with very numerous depressed ulcers; these are commonly serpiginous in character and run into one another with islets of mucous membrane remaining intact between them. In some cases only small depressed circular or oval ulcers filled with white gelatinous mucus are seen. The fibrinous deposit of the acute stage is now absent, and microscopically the floors of the ulcers show fibrous-tissue formation with little tendency to regeneration of the epithelial layers of the mucous membrane. These differences in the distribution of the lesions in the acute and chronic stages respectively are of much practical importance in regulating the treatment.

Symptomatology.—The onset is usually sudden. Abdominal discomfort is quickly followed by severe griping pain with much straining and the passage of blood and mucus, which brings only temporary relief. The frequency of the stools varies greatly from twenty or more to only two or three in the day. The appetite is lost and the tongue furred, but sickness is rare.

Fever is an important feature in the early

acute stage of the disease. The temperature is sometimes remittent, and, in exceptionally severe cases, may be of the continued type for the first few days. More frequently it is intermittent, rising to 101° F. or over. If improvement is taking place the temperature gradually declines by lysis, but even a rise to a little over 99° F. is of significance as an indication of continued activity of the disease. In chronic cases of over a month's duration the temperature is usually normal or subnormal, and only occasionally rises to between 99° and 100° F.

Abdominal symptoms.—Griping pain is common, and is referred to the neighbourhood of the navel. Local abdominal signs are less pronounced than in amœbic dysentery. Usually no thickening of the bowel can be found on palpating the abdomen, although sometimes the sigmoid is contracted and can readily be felt. Tenderness in the course of the large bowel is also comparatively rare in bacillary disease; when present, it is most marked over the sigmoid flexure. In very acute cases only is there much tenderness on palpating the abdomen.

In chronic cases the sigmoid flexure is frequently thickened and contracted, but tenderness is usually absent over it, and is very rare in the cæcal region. Localized peritonitis is unusual in bacillary dysentery, but common in the amœbic disease.

The stools differ widely in character in accordance with the stage and severity of the disease. As a rule mucus is more prominent and copious than blood. In the acute stage the stools sometimes consists of pure mucus resembling the white of an egg, but more frequently it is mixed with blood to a greater or less extent. At first the stools are often composed of pure mucus and blood without any faecal matter, constipation, correctly speaking, being present because the inflamed state of the large bowel prevents the passage of faeces. When improvement takes place, especially under the saline treatment, the blood disappears first and the mucus becomes mixed with loose faecal matter. In milder or more chronic cases blood may not be evident to the naked eye, but can usually be detected with the microscope. On examining the liquid faeces in a thin layer in a glass dish, small pieces of glairy white mucus are seen floating in it, or larger fibrinous masses may be picked out with a strong platinum loop. To form an adequate idea of the progress of dysentery cases Goodeve's method of

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washing the stools is very valuable. Water is poured on each stool, and after the mucus and sloughs have had time to settle, the supernatant fluid containing the faecal matter is gently poured off, the process being repeated until only the pathological products remain. By keeping the washings together the amount of mucus and the character of any sloughs present may be noted daily. It is well to keep a morning stool unwashed so that the amount of blood present can be noted. The daily progress of the case can easily be followed by this means.

The *microscopical characters* of the stools are also of importance. It is not always possible to distinguish clearly between the stools of bacillary and those of amoebic dysentery even by means of the microscope, unless the specific organisms are found. Either the naked-eye or the microscopical method, however, will often enable an experienced physician to form a tolerably accurate opinion. The chief microscopical characters of bacillary dysentery stools are the presence of many columnar epithelial cells derived from the inflamed mucous membrane, and of much fibrin in long, wavy streaks running through the faecal matter. In bacillary dysentery pus cells of the polynuclear type are numerous, and degenerative changes producing badly staining ghost cells common; while in the mucus of amoebic dysentery the cells, which are comparatively few, are mainly mononuclears. The absence of large living active amoebae is an important negative sign, as the repeated absence of these organisms after successive careful examinations (emetine not having been given previously) goes a long way towards excluding amoebic dysentery, and leaves the bacillary form as the most likely remaining condition. For complete confirmation of the diagnosis the isolation of the dysentery bacillus is necessary, but a well-equipped laboratory and an experienced bacteriologist are required for this purpose, and are seldom available in the tropics. Moreover, in chronic cases, even such facilities often fail to separate the few dysentery bacilli from the millions of other organisms. C. J. Martin obtained positive results in 65 per cent. of cases examined in the first five days of the disease, but only in 3-1.5 per cent. from the fifteenth to the fiftieth days.

Complications.—Peritonitis and perforation of the bowel are very much rarer than in the amoebic disease. Hepatitis is equally rare, and so is portal pyæmia from septic infection

through the ulcers. Arthritis, sometimes of an intractable nature, but with a favourable ultimate prognosis, occasionally ensues. In chronic cases anæmia, and œdema of the feet are serious signs.

Diagnosis.—Dysenteries as a class have to be differentiated from other diseases characterized by the passage of blood and mucus from the bowels. These will include *intussusception*, chiefly seen in children, and *cancer* of the sigmoid flexure or rectum in later life. Chronic dysentery is closely simulated by *membranous colitis*. A prolonged history without much constitutional disturbance, and the absence of blood in spite of the occurrence of much stringy mucus, point to the latter affection.

The more difficult differentiation of the bacillary from the amoebic form will be discussed after describing the latter condition.

Prognosis.—In the relatively uncommon acute toxic form with high fever, and especially in its choleraic variety, with copious watery stools and early signs of collapse, the prognosis is grave. When the disease is of moderate severity and comes early under skilled treatment, the prognosis is good as a rule. The later the treatment is commenced the greater is the danger of the disease passing on into the more intractable chronic form. If this happens the prognosis becomes much more serious, for when the mucous membrane of the large bowel has been extensively destroyed and the patient has become emaciated and anæmic, reparative processes are very difficult to bring about.

The **mortality** in epidemic dysentery in Japan was about 30 per cent., whilst in very chronic cases among debilitated Mecca pilgrims at El Tor in Egypt it was over 60 per cent., but in both cases it has been greatly reduced by the serum treatment. In a large Calcutta hospital, where the serum treatment is not available, on account of its cost, the mortality of all forms of dysentery together, among which chronic cases predominate, is about 40 per cent. On the other hand, in early cases as seen in the British Army in India, the death-rate during five years was but 2.93 per cent., and in the Indian Army, including many very mild cases, only 0.51 per cent. In Indian jails, with a larger proportion of chronic cases the figure was 5.29 per cent. in the same period of years. In these figures also all types of dysentery are included, probably many being mild catarrhal affections

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of the large bowel and not caused by the specific bacillus or amœba.

Treatment.—In the acute stage, as soon as the amœbic form of dysentery has been excluded by microscopical examination of the stools in places where that form occurs, no time should be lost in commencing the saline treatment, which is of the greatest possible value at this period. As soon as copious liquid stools are obtained the mucus and blood become greatly diminished, probably by relieving the congestion of the mucous membrane. If this treatment is efficiently carried out before ulceration has taken place, acute cases are frequently cured within a very few days. Sodium sulphate is more palatable and less griping than the magnesium salt. Half an ounce of either in 2 oz. of water, or both combined, may be given as the first dose, followed by 1 dr. of the salt every two hours until free watery stools are obtained; it may then be reduced in frequency to every four hours and continued for three or four days. When all the acute symptoms have subsided completely, and the blood and mucus have disappeared from the stools, bismuth salicylate in 20-gr. doses may be useful. In mild cases the foregoing treatment is all that is required. When much fever and constitutional disturbance are present, anti-dysenteric serum should be resorted to at once in doses of not less than 40 c.c., repeated once or oftener daily until the acute stage is over. It may be given hypodermically or intramuscularly, but if the symptoms are grave should be injected into a vein until decided improvement results. The serum is most useful in the first six to nine days of an attack, but is of little value in the later stages. If the serum has to be administered to a patient who has had an injection more than ten days previously, $\frac{1}{2}$ –1 c.c. should first be injected hypodermically three or four hours before the full dose is given, to lessen the danger of anaphylaxis. At El Tor the best results were obtained with a serum made by injecting horses with strains of bacilli isolated locally, but that of the Lister Institute and those of several well-known firms are efficient. In some cases of dysentery, perchloride of mercury is of value, and 1 gr. each of calomel, opium, and ipecacuanha is recommended by some authorities. A starch-and-opium enema at night is useful to lessen the tenesmus and frequency of the stools in order to obtain sleep. Emetine and ipecacuanha are useless in bacillary dysentery.

In chronic cases salines may do more harm than good, and, usually, little benefit is obtained from drugs by the mouth, as they are rapidly expelled after they have reached the ulcers in the lower part of the large bowel. It is in this stage that medicated enemata find their place in the treatment. Silver nitrate in strengths of 1 gr., gradually increased to 5 or even 10 gr., to the ounce has long held the first place among such remedies, but the writer has found that albargin (silver gelatose) and nargol (silver nucleate), in the strength of 1 gr. to the ounce, are often more efficacious because they escape precipitation by chlorides and albumin while they are less painful. Thus, in the presence of albuminous fluids, they are much more active against the dysentery bacillus than is silver nitrate. Copper sulphate, in a strength of 1 gr. to the ounce, is sometimes useful. Solutions of boric acid have little or no antiseptic effect on the dysentery bacillus, and merely act by cleansing the bowel, for which normal saline may also be used, while solutions of permanganate of potash have a cleansing effect and neutralize dysentery toxins. A 1-per-cent. solution of sodium bicarbonate may be used for dissolving the mucus before giving stronger injections. From $1\frac{1}{2}$ to 2 pints or more of the selected solution should be run in slowly through a funnel held only a foot or so above the level of the patient and connected with a soft catheter passed some 8 in. into the rectum, the patient being in the semi-prone position; the injection should be repeated every morning.

Fresh ripe bael fruit is sometimes of service. Ispaghula is also used as an emulcent, and innumerable other drugs have been advised from time to time.

The serum treatment has been found to be of great service in chronic, as well as in acute, cases at El Tor. Vaccines made from killed Shiga bacilli were used by their discoverer as a prophylactic measure, and by Forster in India in the treatment of chronic dysentery, with good results in some cases. The writer has found that sensitized dysentery vaccines are safer and of considerable value. Operative measures are rarely required, and appendicostomy is now rarely performed. In severe chronic cases, as a last resource, cœcostomy has occasionally proved of service.

Diet.—In the acute stage of dysentery citrated milk or whey and albumin water should first be given. Much caution is required in adding to the diet, for if relapses occur there

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is a danger of the disease becoming chronic. In the later stages a somewhat more liberal diet must be given to keep up the patient's strength, farinaceous and other easily digestible foods being added to the milk. Sour milk has been recommended, but usually fails to be of special service.

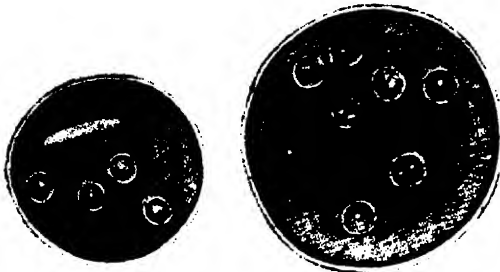
AMOEBIIC DYSENTERY

An ulcerative condition affecting chiefly the submucous and mucous coats of the large bowel, caused by the *Entamoeba histolytica*, and producing symptoms of dysentery or diarrhoea, often complicated by hepatitis or abscess of the liver.

Etiology.—There has been much controversy regarding the pathogenicity of the

quired to detect the difference in the cysts. For practical purposes the following points of distinction, as seen in the examination of fresh portions of stools, have often to be relied on. The *E. coli* presents uniformly grey granular protoplasm, the ectoplasm not being differentiated from the endoplasm; the nucleus has a well-marked ring of chromatin; and very rarely are any red corpuscles found within the organism. On the other hand, the *E. histolytica* has pseudopodia of clear ectoplasm well defined from the central granular endoplasm, and the organism often contains numerous red corpuscles. The nucleus has only a thin rim of chromatin, and is difficult to see in unstained specimens. Thus, if fairly numerous, large active amœbæ with clear ectoplasm, and some of them containing red corpuscles, are seen, they may safely be regarded as pathogenic. The organisms can readily be seen with a $\frac{1}{2}$ -in. lens in a piece of mucus spread thinly under a cover-glass on a warmed slide. The examination is therefore a simple one, and should never be neglected in cases of dysentery and diarrhoea where amœbic disease is prevalent. By adding a drop of a 1-per-cent. watery solution of methylene-blue to the mucus the organisms stand out more distinctly as nearly clear bodies among the stained mucus and faecal matter. This is a useful method when they are scanty. Under favourable conditions entamœbæ encyst, acquiring a double outline and losing their mobility, while their nuclei can be made visible with a strong iodine solution. In this form they may survive for a time outside the body and may gain access to new hosts through water and food. Fresh infections generally occur during the rainy season in hot, damp weather, which is favourable to the survival of the encysted forms outside the body, but relapses take place at any time of the year.

E. histolytica and *E. coli* usually measure 18-40 microns, but in addition a smaller harmless variety, *E. nana*, with a diameter of 6-12 microns, occurs not rarely in human stools; it resembles the non-pathogenic *E. coli* in its sluggish movement and the absence of red corpuscles from its protoplasm. In stained specimens it has a characteristic nucleus with one large mass of chromatin connected by fine threads with one or more smaller masses, while its cysts are oval, with four small nuclei. Yet another occasional human intestinal amœba has been described under the name of *Iodamoeba bütschlii*, resembling a small *E. coli*, with a



C. Dobell, del.

Fig. 21. — Fully developed cyst of *E. histolytica*, with four nuclei. $\times 2,000$.

Fig. 22. — Mature cyst of *E. coli*, with eight nuclei. $\times 2,000$.

(From Report No. 51 of the Medical Research Council, by permission of H.M. Stationery Office.)

amœbæ met with in the human intestine, but the following points appear to be now clearly established, largely as the result of the work of Walker. The *Entamoeba histolytica* is the only pathogenic form, that described as *E. tetragena* being only another stage of the same organism. In addition, a harmless saprophytic organism, the *E. coli*, may be found in the large bowel, but is incapable of producing disease. The human intestinal amœbæ have not yet been cultivated outside the human body, such organisms as can be grown from stools on artificial media being derived from the cysts of harmless air- or water-borne amœbæ, which have passed unchanged through the intestinal tract. Only in the cyst stage can these two forms of human entamœbæ be easily distinguished, the fully developed pathogenic *E. histolytica* having four nuclei, and the harmless *E. coli* eight (Figs. 21, 22). Examination of stained specimens by a skilled observer is re-

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diameter usually between 9 and 13 microns ; the stained nucleus shows a large central mass of chromatin, while its cysts are of irregular shape with a single nucleus, and its protoplasm contains a large glycogen body staining brown with iodine.

Pathology.—The naked-eye changes in the large gut are characteristic, although they differ widely in degree, varying from a rapidly fatal gangrene of the whole thickness of the bowel-wall to the few small latent ulcers, unaccompanied by active bowel symptoms, so often seen in a patient who has died of hepatic complications. The primary and most important seat of the disease is the submucous coat, to which the organisms penetrate through the tube glands, and produce such extensive gelatinous and cellular infiltration that the submucous layer may be thicker than the whole of the rest of the bowel-wall. The mucous membrane over the affected patches then sloughs away, leaving a raised ulcer with a tawny yellow base and congested edge, clearly defined from the normal relatively depressed healthy mucous membrane around it. These ulcers are at first rounded or oval, but in acute cases may spread and involve the whole circumference of the bowel for several inches, still retaining, however, abruptly rising edges where they meet the healthy mucous membrane. Attached to the edges of such large ulcers, grey or black sloughs of the mucous membrane may be seen. The muscular coat is also involved in the inflammatory process, and portions of the peritoneum may be affected without actual perforation. Such cases are rapidly fatal unless correctly treated.

The lesions differ from those of the bacillary disease in that the ulcers are raised yellow patches of exposed thickened submucous coat scattered over an otherwise healthy mucous membrane. In the bacillary disease there is general thickening of the mucous membrane, the ulcers being irregular and depressed and frequently running into each other.

In very chronic cases of amoebic dysentery extensive ulcers, slightly depressed and partially healed, may also be found. Marked pigmentation may be seen both around them and at the sites of others which have healed completely. Sometimes every stage of the pathological lesions may be seen in different portions of the same large gut.

The distribution of the lesions is different also in the two forms of dysentery. In the amoebic form they are generally most numer-

ous in the cæcum and upper half of the large bowel, and, although the sigmoid flexure also is often affected, both it and the rectum may escape, especially in subacute and chronic cases. In the latent form without severe bowel symptoms the ulcers are limited usually to the cæcum and ascending colon. Since the disease is then unrecognized, serious liver complications often ensue in an insidious manner.

The *microscopical characters* are best studied in the small recent circular ulcers. At this stage the submucous coat shows a thick gelatinous layer with scanty mononuclear-celled infiltration, in which numerous amoebæ are readily found. The tube glands of the mucous coat are fairly healthy close to the edge of the ulcer, since the mucous membrane is not primarily affected. In more severe and advanced cases the process has invaded the muscular coat and even reached the peritoneum. Amoebæ may be found also in the small veins of the submucous coat, through which they may reach the liver.

The blood-changes are important. In the acute type, leucocytosis is nearly always present, and is often of a high degree. There may be from 25,000 to 50,000 leucocytes per c.mm. or more ; this is always a grave sign. In mild and chronic cases with anæmia either an actual or a relative leucocytosis (i.e. more than one white cell to 500 red) generally occurs. This is a point of importance in distinguishing the condition from chronic bacillary dysentery, in which the leucocytes are rarely much increased. In acute cases of amoebic dysentery the polynuclear cells may be in excess, whilst in chronic cases their percentage may even be diminished.

Symptomatology.—Few diseases run so variable a course as amoebic dysentery, or, more correctly, amoebic colitis—for there may be diarrhoea only, the ordinary signs of dysentery being absent. The symptoms of most cases do not differ sufficiently from those of the bacillary form to enable a clinical differentiation to be made with certainty without the aid of the microscope. It occurs in all degrees of severity, from the acute fulminating to the chronic type. The former terminates within a few days in patches of gangrene involving the whole thickness of the bowel-wall, with local peritonitis over the sloughing areas, perhaps accompanied by perforation ; the latter may be characterized by relapses recurring over a number of years, or the lesions may be restricted to a few latent ulcers high up in the large gut only producing occasional slight

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diarrhoea, but liable to lead to fatal hepatic complications. Moreover, chronic diarrhoea, without obvious blood or mucus in the stools, may result from amœbic infection and may terminate fatally, the true nature of the disease being unrecognized during life. Indeed, in a series of post-mortem examinations on the bodies of patients who had died from amœbic disease of the bowel in Calcutta, no less than 40 per cent. were in cases which were not diagnosed as dysentery during life.

In the more acute forms of amœbic dysentery the stools are usually very numerous, often exceeding twenty in a day. As a rule there is not much straining, since the lower part of the large bowel is less affected than the upper portions; but defæcation is preceded by griping pains in the abdomen. The stools consist either of pure blood and mucus, or of rosy masses mixed with faecal matter. Owing to the sloughing of the deep layers of the bowel-wall, copious and dangerous hæmorrhages may also occur. Usually the mucus is less intimately mixed with the faecal matter than it is in the milder forms of bacillary dysentery, but this is not a constant difference. In more chronic cases blood may not be visible to the naked eye, although usually it can be found microscopically. Obvious mucus may also be absent, only loose faecal matter being passed. Such cases are very liable to be overlooked unless the stools are subjected to a microscopical examination.

The abdominal signs are of great importance. In very acute cases the greatly thickened colon can be felt through the abdominal wall as a rounded or elongated mass, and is often tender from involvement of the peritoneal coat. This is always a serious symptom. Such tumour-like masses are found most frequently in the position of the cæcum and ascending colon, but may occur in relation to any part of the large bowel. In cases of moderate severity, slight thickening and tenderness on deep pressure are often met with, especially in the cæcal region, and lend considerable support to the diagnosis of amœbic, as opposed to bacillary dysentery.

The onset of the disease is usually sudden, but in mild cases may be insidious and commence with slight diarrhoea. The constitutional symptoms are very variable, but are usually less severe than in the acute bacillary form. Fever is absent in about half of the cases. It is of the remittent type in severe attacks, moderate in degree and intermittent

for the first few days in acute and subacute attacks, and commonly absent in chronic cases.

Complications.—In acute cases local or perforative peritonitis is the most serious complication. Occasionally retrocæcal or retrocolic abscesses may result; generally they are on the right side and are liable to be mistaken for those of appendicitis. Rarely, there may be perforation of the appendix itself, due to amœbic ulceration.

In chronic or latent cases, amœbic hepatitis progressing insidiously into liver abscess, frequently occurs (*see* TROPICAL ABSCESS). Secondary abscesses in the spleen, joints, or brain have also been recorded.

Diagnosis.—Although the features already mentioned often permit a correct differentiation between amœbic and bacillary dysentery to be made, microscopical examination of the stools for amœbæ should always be carried out at the earliest moment before specific treatment is commenced, as the latter may cause the organisms to disappear from the stools within twenty-four hours, and some doubt may remain as to the correct diagnosis. It is true that the rapid improvement under emetine is practically diagnostic of the amœbic disease, still it is more satisfactory to ascertain definitely the presence or absence of the causative organism before commencing treatment, especially as it only takes a few minutes to make the necessary simple microscopical examination of a stool.

In countries where amœbic disease is prevalent the stools of chronic diarrhoea cases should always be examined for amœbæ, as obvious blood and mucus may long be absent from the stools even when large numbers of pathogenic amœbæ are present. Amœbic dysentery has a greater tendency to relapse than the bacillary form and to cause repeated attacks extending over several years if not treated with adequate doses of emetine.

Such causes of blood and mucus in the stools as cancer in older people and intussusception in children must always be borne in mind.

Prognosis.—Up to 1912, amœbic dysentery was one of the most intractable of diseases, and frequently proved fatal either by exhaustion or by means of its complications. The prognosis has, however, been materially altered by the introduction of hypodermic injections of emetine hydrochloride by the writer. The prognosis now depends entirely on the stage of the disease in which the patient comes under skilled treatment. A few cases

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are still seen for the first time when the process is so acute that gangrene of the whole thickness of the bowel-wall and local peritonitis, with or without perforation, have already occurred. Such cases commonly terminate fatally within two or three days, before there is time for treatment to be of much avail. Occasionally the acute symptoms may subside under treatment, but the wall of the large gut has been destroyed so extensively that ultimate recovery is impossible and perforative peritonitis or a postcolic abscess may result. Apart from such cases and those already complicated by extensive destruction of the liver, the prognosis as regards the immediate future is very good, although, even under emetine treatment, relapses occasionally occur after some weeks or months, if the injections are left off too soon after the symptoms have disappeared, and a few cases prove intractable even to emetine. Hepatic complications, especially if they have been allowed to drift on into amoebic abscess of the liver, add greatly to the gravity of the disease.

Mortality.—Under the old ipecacuanha treatment, even with large doses, the mortality in Calcutta hospitals, where cases of a serious class are seen, was from 30 to 40 per cent. Under the new emetine treatment the mortality in my wards fell to about one-fourth of the former figure, and most of the fatal cases died within the first two or three days after being admitted in a hopeless condition.

Treatment.—Soon after the writer had demonstrated, in 1901, the frequent occurrence of amoebic dysentery in India, he came to the conclusion that ipecacuanha, which had long been given in large doses in all forms of dysentery, was effective in the amoebic form only—a view which Sir Patrick Manson long held. Finding that even ipecacuanha often failed, and remembering Vedder's experiments on the destructive effect of emetine on non-pathogenic amoebæ, the writer tested its soluble salts on pathogenic amoebæ in dysenteric stools, and found that high dilutions had a very rapid and marked effect on their activity. By injecting the emetine salts hypodermically, he proved them to be truly specific against the disease, and his observations have now been so fully confirmed in all parts of the world where the disease occurs, that the new treatment has practically displaced all the numerous remedies formerly given by the mouth or as medicated enemata.

The most convenient salt is the hydrochloride of emetine, which is more soluble than the hydrobromide. Cephaeline salts are much less efficacious than those of emetine. A mixture of both may be used in somewhat larger doses than the emetine alone, but possesses no advantage except in cheapness. Although the great drawback to the administration of large doses of ipecacuanha by the mouth was the severe nausea and sickness it produced, emetine, even in 1-gr. doses (the equivalent of 90 gr. of ipecacuanha), rarely produces nausea, and scarcely ever vomiting. In adults, 1 gr. should be injected daily for eight to twelve days, as shorter courses are often followed by relapses at a later date. The injections may advantageously be supplemented by subsequent oral administration of full doses of ipecacuanha in recurring cases. In the very acute cases, with a thickened and tender colon, more vigorous treatment is necessary. A 1-gr. dose, dissolved in 10 c.c. of sterile water or normal saline, should at once be very slowly injected intravenously, and a similar dose should be given subcutaneously twice the same day, and repeated daily until the acute stage is passed. It is surprising how rapidly a tumour-like mass of thickened bowel will clear up under this treatment if fatal damage has not already been done. In adults, 2 gr. a day may safely be injected hypodermically, and a case has been reported in which 4 gr. were given at one time before the amoebæ disappeared from the stools. Children bear the drug well, and $\frac{1}{4}$ -gr. doses may be administered to those over 10 years, $\frac{1}{2}$ -gr. doses to those between 5 and 10. The injection may conveniently be made in the upper arm about the insertion of the deltoid or just below the clavicle, but not in the forearm. It is best to dissolve a 1-gr. dose in not less than 20 min. of sterile water, as the pain is less than after the use of more concentrated solutions. The solution may be boiled for a short time without harm. There is no serious disease in which the treatment is more definitely specific than it is in amoebic dysentery if the soluble salts of emetine are employed. The salt may also be given by the mouth in pills coated with keratin or salol, but although they are better than the bulky ipecacuanha, they often cause nausea and sickness. This method of administration acts much less rapidly than the hypodermic method, and consequently is only indicated when the latter is not available. In the few cases which relapse or become cyst-carriers in spite of the above

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treatment, emetine bismuth iodide in 3-gr. doses orally for twelve consecutive days is the most efficient treatment, although the drug is rather irritant to the bowel.

The bowels should be kept open, preferably by castor-oil emulsion in appropriate doses. This prevents the accumulation of fecal matter in the ulcerated large bowel, and counteracts the tendency of emetine to produce constipation. Medicated enemata, which formerly were so largely used and continued for months at a time, are no longer required; in acute cases they are dangerous and may cause perforation of the bowel.

It is unnecessary to deal at length with other methods of treatment. Deeks has reported very good results in Panama from the administration of bismuth subnitrate (180 gr. in a tumblerful of water every three hours, day and night), but he has recently confirmed the specific effects of the emetine treatment.

Diet.—A fluid diet consisting mainly of milk is advisable for several days, although it is surprising how soon the ulcers heal under the treatment by emetine, lesions in the rectum as large as an inch in diameter disappearing within a few days. As the dysenteric symptoms usually clear up within three days, farinaceous food can soon be given. This can quickly be supplemented by other easily digested food, since convalescence is very soon established.

LEONARD ROGERS.

DYSIDROSIS (see POMPHOLYX).

DYSMENORRHEA.—It is estimated that 65 per cent. of all women suffer from pain during menstruation, not of course necessitating rest in bed, and at least 15 per cent. of all married women are believed to spend a part of each period in bed. Some people show a more ready toleration of pain than others, and even in the same person this toleration varies with the degree of physical fitness.

The pain arises in three ways. One is a faulty uterine mechanism by which the uterine contractions, which work towards expelling the contents, fail to secure correlative dilatation of the cervix, and consequently painful and more violent contractions ensue; a second is the undue size of the material to be discharged, so that unusual contractions are invoked, as when a clot forms, or a fibroid occupies certain positions. A third factor is pelvic congestion, affecting not only the uterus but the pelvis generally.

Three clinical types of dysmenorrhœa are now generally recognized—*Spasmodic*, *Membranous*, and *Congestive*.

The description of an *ovarian* type of dysmenorrhœa has now been abandoned, for from clinical experience it has been possible to demonstrate a pathological change underlying all such pain.

Formerly, too, an *obstructive* form of dysmenorrhœa was described, but it is doubtful how far bending or kinking of the uterus causes difficulty in expulsion. Thus, in what is described as congestive dysmenorrhœa with a retroflexed uterus, the pain has not the character of the spasms of uterine contractions, although relief is complete on correcting the displacement.

Occasionally, however, a small fibroid in the cervix is found in dysmenorrhœa, but even in these instances it is the greater uterine contractions involved that account for the pain, firstly in order to force the fluid beyond the inert mass, and secondly in order to expel the mass itself.

In membranous dysmenorrhœa the formation of a partial or complete endometrial cast and the efforts necessary for its expulsion are the cause of the pain.

It thus follows that only the spasmodic variety is, strictly speaking, a true dysmenorrhœa, that the congestive type is merely pain present or exacerbated at the period but bearing no direct relation to the menstrual process, and that the membranous form is more correctly an endometritis characterized by dysmenorrhœa.

SPASMODIC DYSMENORRHEA

This form occurs commonly in the young, at or within a year or two of nubility, and may last throughout menstrual life. It is not associated with any disease in other parts, although of late years it has been suggested that the internal secretions may be held responsible, either by excess or insufficiency. There are also varieties in which a poor circulatory system is much in evidence.

Symptomatology.—The characteristic symptom is pain associated with or but slightly antecedent to the establishment of the menstrual flow. It is centred in the hypogastric region, and radiates thence, is colicky in character and is intermittent, the spasms varying in intensity and duration. In its most severe form it may cause vomiting, general perspiration, fainting, and collapse. After

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from twelve to twenty-four hours the pains lessen in severity, and a freer discharge occurs.

The pain and exhaustion often necessitate remaining in bed, but recumbency does not relieve the pain.

On pelvic examination certain abnormal conditions of the uterus are sometimes encountered, which may explain the origin of the pain. A *bicornuate uterus* is frequently associated with a most severe form, which nothing short of hysterectomy will relieve. An *infantile uterus* may be detected, or a uterus which is anteflexed, especially if the cervix is large in comparison with the body or if the cervix is conical. In these conditions the dysmenorrhœa is rather intractable; it is probable that the incomplete development of the uterine musculature, and with it a resulting faulty "polarity," offers the best explanation of the pain. If some definite pathological condition, such as a small fibroid, is discovered, it may be held to account for the pain and treated accordingly.

In the majority of cases, however, nothing abnormal will be detected in the pelvis, and the spasmodic pains appear to arise from excessive contractions owing to the failure of the cervix to dilate as in normal periods. An alternative explanation may be found in the nature of the contents to be expelled. Thus recent investigations tend to show that the uterine blood escaping from the endometrial vessels on dehiscence of the necrotic endometrium at once coagulates as in ordinary hæmorrhage, but that an enzyme, present in the endometrium, acts on the coagulated matter and causes its disintegration.

It is evident, then, that spasmodic dysmenorrhœa is generally due either to a faulty uterine development or to faulty uterine metabolic processes, and consequently it is not surprising that sterility should be noted in about 50 per cent. of all cases and should be intractable in 25 per cent. of cases. A case of dysmenorrhœa in which pregnancy occurs may be regarded as cured. Unfortunately, in the worst cases sterility is the rule.

The periodicity of the menses is perfectly rhythmical, the loss is scanty, especially in severe types, and lasts but a few days. Where a bicornuate or ill-developed uterus exists, long periods of amenorrhœa may occur.

Treatment.—Free saline purging a day or two before the period, and the application of frequently changed hot fomentations to the

hypogastrium, with the administration of a sedative of the nature of bromide or aspirin, are the palliative measures offering the best chance of relief. In very severe cases a large dose of bromides and chloral may be given in the form of an enema.

Alcohol, opium, and morphia are best excluded, as alcoholism and drug habit have so frequently owed their origin to the temporary relief which these drugs afford. Alcohol particularly should be condemned, while morphia, if it is necessary, should be given in a rectal suppository. Numerous drugs have been used at one time or other, but none appears to have any effect except those which possess analgesic and sedative properties; these produce temporary relief.

Prolonged treatment with guaiacum was at one time popular, but has fallen into disuse. Conditions tending to improve the general health should be favoured.

The explanation offered to account for the pain in this form indicates that the treatment most likely to effect a cure is *dilatation of the cervix*, especially in those cases in which no gross pathological lesion is found on examination. The effect of dilatation is to stretch and rupture the circularly arranged fibres so that uterine contractions become capable of expelling the contents without the necessity for tetanic, cramp-like pains. In dilating the cervix, it is essential to stretch the internal os to the point at which it is felt to give; this is generally between sizes 12 and 15 of the uterine dilators. Failure to do this accounts for a number of unsuccessful results.

The procedure recommended is the following: In the first instance, and in the absence of palpable uterine abnormality, to perform dilatation, which may be undertaken: (a) As a curative measure, that is to say, in order to permit menstruation which is not very painful; it is successful in about 30 per cent. of cases, and the chance of success is enhanced if there is a likelihood of pregnancy shortly supervening. (b) To effect relief; this follows in about 40 per cent. of cases, and lasts from six months to three years, when the operation requires to be repeated, or some more radical measures adopted.

Next, in those cases—a considerable number—in which the dilatation fails, after displacing the bladder from its anterior surface, the cervix is split well up into the lower segment of the uterus (*vaginal hysterotomy*) and the vaginal wall stitched to the inner surface of the split

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cervix. It should be remembered that sterility is common after this procedure.

Lastly, in the most severe and intractable cases a *hysterectomy* offers the only chance of permanent relief. *Bilateral ovariectomy* has been performed, but offers no advantages over hysterectomy and has the disadvantage of the artificial menopause.

CONGESTIVE DYSMENORRHEA

Dysmenorrhœa of the congestive form is characterized by pelvic pain which is more or less constant, slight or pronounced, but is exacerbated a few days to a few hours before the onset of the period. The persistent pain is due to pelvic inflammation from one cause or another. When to this is added the increased tension and congestion incidental to the premenstrual flux a decided exacerbation of the pain occurs, to be followed by a corresponding relief with the establishment of menstruation.

Etiology.—Congestive dysmenorrhœa is seldom primary, except in the few cases in which retroflexion has occurred during childhood. The conditions giving rise to it are chronic inflammatory affections of the ovaries and tubes, especially if they are prolapsed into the pelvis; backward displacements of the uterus, in which a mild form of metritis exists; pelvic tumours of whatever nature, if impacted in the pelvis; peritoneal adhesions from a previous appendicitis or other cause; varicocele of the broad ligaments; and finally simple prolapse of an appendage into the pelvis. It may follow sudden strain in single women, or be the result of pelvic or puerperal infection in the married. It may therefore appear at any time during menstrual life.

Symptomatology.—The amount of pain depends upon the cause and its position. Thus a retroverted metritic uterus is a fertile source of very severe premenstrual exaggeration of the pelvic pain; a small prolapsed ovary in which the circulatory return is disturbed causes distressing symptoms, while a large inflammatory swelling or new growth with good circulation produces a pain which is mild in comparison. The pain may be felt in the back, through a sacro-iliac region, through the hip to one or other side, and finally low in the sacrum or pelvis, the site depending upon the position and side on which the pathological condition exists. It is seldom of the agonizing, acute kind met with in spasmodic dysmenorrhœa, is relieved by recumbency which facili-

tates the circulatory return from the pelvis, and is eased by the application of warmth to the back. It is felt as a splitting backache, a bearing down, or a dull, incessant ache, and is continuous and not intermittent as in the spasmodic type. Headache is common.

The periods usually last for a day or two longer than normal, the menstrual flow being excessive in quantity and succeeded by a mucous discharge of a few days' duration.

Treatment.—This form can be cured completely. Nothing is more satisfactory than the results obtained by certain surgical measures directed towards the cure of displacements and the extirpation of chronically inflamed appendages. Pessaries are now discarded in favour of curative procedures. Provided a correct diagnosis of the cause is made, relief may be assured. During the periods, it may happen that some palliative treatment is necessary, and the following measures will be found beneficial, viz. (a) the application of heat, in the form of hot-water bottles to the seat of pain, (b) a hot saline douche or rectal injection. (c) glycerin and ichthyol suppository, (d) aspirin and bromides per rectum.

MEMBRANOUS DYSMENORRHEA

Membranous dysmenorrhœa is characterized by the passage of pieces of membrane and, less commonly, of a complete membranous cast of the uterine lining. The pain is similar to that of the spasmodic form, being intermittent, severe, and cramp-like; it is only eased by the expulsion of a strip of membrane. It is located in the hypogastric region, and does not necessarily recur at each period; it may, indeed, only occur very occasionally, as it depends both on the causal factor and on the solidity, or degree of degeneration and disintegration, of the uterine cast. In a characteristic case there is an excessive loss followed by the spasmodic pains, which begin about the second day, increase in intensity, and only end with the passage of the membrane.

The **pathology** is obscure. It is usual to regard the cases as instances of a monthly abortion, on account of the decided enlargement of the stroma cells of the endometrium which, in well-preserved specimens, gives them many of the characters of decidual cells. No chorionic villi, syncytium, or foetal elements have, however, been demonstrated. A case recorded by Blair Bell, which recurred after bilateral salpingectomy, would appear to rule out this explanation. A similar decidua-like

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enlargement of the endometrial stroma is encountered in the neighbourhood of small tuberculous foci of the uterus; and, further, in Blair Bell's case referred to above, if the patient was kept in hospital, the membranous cast was not formed, but when she cohabited with her husband the dysmenorrhœa recurred, even in the absence of the ovaries. Hence it is to be assumed that certain stimulants are capable of provoking the formation of a decidua-like enlargement of the endometrial tissue, which is apt to be shed *en masse* or in a partial state of disintegration only, and that among them are included the tubercle bacillus, spermatozoa, and possibly others.

The subjects of this variety of dysmenorrhœa are found most commonly among the married, and they are almost invariably sterile. The **prognosis** is unfavourable, as when the condition is once established recurrence is the rule.

Treatment.—If the dysmenorrhœa is crippling in its severity, certain and effective relief is only to be obtained by *hysterectomy*. Dilatation of the uterus, curetting and direct applications are still usually tried in the first instance, but are seldom successful, except in the mildest forms, in which the membrane is passed in small strips and is necrotic.

BRYDEN GLENDINING.

DYSPAREUNIA.—A condition of painful or difficult coitus, and thus a symptom of one of many local lesions. In dealing with a case of dyspareunia it is important to notice the age of the patient, the duration of the pain, and the relation of its onset to marriage; also the site of the pain should be inquired into, and its time-relations to the sexual act. Thus it may be described at the "inlet," commencing with the act, or "deeply inside," at its worst during and after the orgasm. In the one case the seat of the trouble is almost certainly the vulva or ostium vagina; in the other, the uterus or the appendages are probably responsible.

Etiology.—The causes are chiefly organic and local, and may be thus enumerated:

1. *Abnormalities of the hymen.*—It may be imperforate, or impenetrable, having a small orifice surrounded by a tight and unyielding edge. The gentlest insertion of the finger causes great pain. Sometimes the hymeneal fissures consequent upon recent rupture have not been allowed to heal, and appear as chronic and exceedingly tender fissured ulcers between the carunculæ myrtiliformes, which in consequence become tender and painful.

2. *Inflammatory conditions of the vulva.*—Acute and chronic vulvitis, vulval fissures which may have resulted from scratching in cases of pruritus, chronic shallow gonorrhœa ulcers, and inflammation of Bartholin's duct and gland.

3. *Urethral caruncles.*

4. The *vagina* is seldom *per se* the cause of dyspareunia. Chronic vaginitis is rare, but very intractable, and when present will cause painful coitus of an extreme degree. As the physical signs are not very obvious, this condition should be sought carefully with a Ferguson's speculum and a good light.

5. Some *secondary contraction of the vaginal orifice* is normal at the menopause, and may cause slight pain, but it occurs in a marked degree in *kraurosis vulvæ*—a disease which is liable to appear at, or just after, the menopause.

6. *Leukoplakia vulvæ*, when it has reached the stage of fissures.

7. A *vaginal cyst* may cause difficulty without much pain. It is usually found low down under the anterior wall, but occasionally occurs high up in the lateral fornix.

8. *Congenital hypertrophic elongation of the cervix.*

9. Certain cases of *retroversion of the uterus*, in which the displacement is fixed or impossible to replace, and especially when enlarged and associated with endometritis. The uterus in this condition is often very tender on vaginal examination, the merest pressure on the fundus, or the slightest attempt to move the cervix, causing pain. Simple backward displacement which is quite mobile and not at all tender should not be considered as a cause of dyspareunia.

10. *Chronic fibrous parametritis* as a result of former parturition injury, together with a tender scar in the vault of the vagina and fixity of the cervix, may cause dyspareunia. In these cases there is considerable pain on moving the cervix at clinical examination.

11. *Chronic salpingo-oöphoritis, with pelvic peritonitis.*

Another group of cases presents no physical signs of organic lesions. The patients are, as a rule, young recently married subjects of an hysterical tendency, and present all degrees of dyspareunia, from mere cold distaste of the sexual act to the most violent repugnance coupled with vaginismus. This condition is one of spasm of the superficial muscles of the perineum and the adductors

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of the thigh, usually with a varying degree of opisthotonos. The slightest attempt at coitus or vaginal examination will induce the spasm. Sexual frigidity is not necessarily found with either painful or difficult coitus, nor is it associated with sterility. It frequently disappears after the first child is born. In rare cases total ignorance of the sexual act is the cause of the trouble.

Treatment.—By a most careful local examination the causal local lesion must be found and dealt with. It is usually easy to treat, and a rapid cure results. Exceptional conditions are cases of chronic vaginitis, which is often extremely refractory, and of uterine and tubal inflammation, which frequently require operation. Dyspareunia due to kraurosis vulvæ is almost incurable, as the contraction of the vaginal orifice is caused by a permanent laying down of fibrous tissue. It is not often that the practitioner is called upon to treat this condition for the symptom of dyspareunia, owing to the time of life at which it occurs. The best palliative remedies are ointments containing cocaine or menthol, but they do not give much relief. Congenital contraction of the vaginal orifice or an impenetrable hymen may easily be cured by excising the hymen and stitching the mucocutaneous edges, followed by backward division of the perineum for about $\frac{1}{2}$ – $\frac{3}{4}$ in., and suture of the wound thus formed in a horizontal direction. The ostium is thereby permanently widened. After the operation it is necessary to prohibit sexual intercourse for at least six weeks.

Vaginal spasm is usually extremely difficult to treat successfully. The patient should be anæsthetized and the vaginal orifice forcibly dilated, with removal of the hymen if necessary. During the ensuing few weeks glass dilators should be passed each day and retained *in situ* for an hour or so. As this condition is largely psychical and hysterical, the foregoing treatment often fails. The patient may then be treated by suggestion.

A. W. BOURNE.

DYSPEPSIA (see GASTRITIS, CHRONIC; STOMACH, FUNCTIONAL DISORDERS OF; DIARRHEAL DISORDERS OF INFANTS).

DYSPHAGIA (see SWALLOWING, DISTURBANCES OF).

DYSPITUITARISM (see PITUITARY GLAND, AFFECTIONS OF).

DYSPNŒA

DYSPNŒA (Difficult Breathing). — Although in its most correct interpretation "dyspnœa" denotes difficulty in the respiratory act, it is convenient to consider under this title other variations from normal breathing. Such are "hyperpnœa," or abnormally deep breathing, and "tachypnœa," or rapid breathing, in both of which, though the act is altered, there is no real difficulty in its performance. The time-honoured division of dyspnœa into "subjective" and "objective" is of little practical use; by "subjective dyspnœa" is meant merely a sense of "shortness of breath," and by "objective dyspnœa" a difficulty in breathing the signs of which are patent to the observer. A survey of the factors concerned in normal respiration suggests a means of classifying dyspnœa, but the results are not very satisfactory, since dyspnœa arises in each disease not by a defect in one of the factors of respiration, but by an alteration of many of them. Thus, in pneumonia, dyspnœa results from a combination of many effects produced by that disease: the lung itself is partly out of action by consolidation and by congestion in its vessels, which may be enhanced by right-heart failure; breathing is made shallow and difficult by the accompanying painful pleurisy, while the increased content of the CO₂ in the blood, and probably also the presence in the latter of the toxin of the disease, increase the respirations by their actions on the respiratory centre in the medulla. In every variety of breathlessness in which there is imperfect oxygenation of the blood the respiratory centre plays its part in the production of the syndrome which we term dyspnœa. An endeavour is made in this article to classify it etiologically as far as possible, though with a full realization that its explanation is more complex than such a classification would seem to imply.

1. **Increased frequency of respiration (tachypnœa).**—This is the commonest alteration, and is the natural response to any difficulty in aeration of the blood. It occurs normally as the result of exercise or exertion and as part of the physical manifestations of fear, anxiety, or nervousness. Of disease it is a very usual attribute, and is met with under the following conditions:—

(1) *Diminution in the amount of lung tissue available for aeration*, as in phthisis, pneumonia, empyœma, fibrosis, infarction, œdema, pulmonary collapse, new growth, and compression

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from pleural effusion, pneumothorax, pleural neoplasms or mediastinal tumours.

(2) *Stagnation in the pulmonary circulation*, as in heart failure, whether due to heart disease or secondary to other causes.

(3) *Impediment to air-entry*, as in nasal or naso-pharyngeal obstruction—spurs, enlarged tonsils, adenoids, retropharyngeal abscess; in laryngeal obstruction—diphtheria, œdema, tuberculosis, new growth; in bronchial obstruction—bronchitis, asthma; and as the result of partial closure of the air-passages by pressure from without, as by tumours, glands, or aneurysm, or by the presence of a foreign body within.

(4) *When breathing is painful and consequently shallow*, as in pericarditis, mediastinitis, intercostal neuralgia, muscular rheumatism, and pleurisy, especially if diaphragmatic.

(5) *Fixation of the chest*, as in old age, emphysema, osteitis deformans and osteoarthritis of the costo-vertebral joints, and by pressure in a crowd.

(6) *Paralysis or paresis of inspiratory muscles*, the result of lesions of the spinal cord, or of peripheral neuritis, particularly post-diphtheritic and beriberi, or reflexly, in association with abdominal operations or acute peritonitis. Another cause is interference with the movements of the diaphragm by acute dilatation of the stomach, flatulence, a tense ascites, or a large ovarian cyst.

(7) *Defects of the blood*, either a reduction of hæmoglobin as in anæmia and after hæmorrhage, or fixation of the hæmoglobin as in cyanide, coal-gas, or carbon monoxide poisoning.

(8) *Alteration in atmospheric pressure*, either a reduction such as is encountered in mountaineering and flying, or increase as in diving and tunnelling.

(9) *Alteration in the composition of the inspired air*, as by the addition of irritating gases, such as ammonia, chlorine, and the gases of war.

(10) *Excitation of the respiratory centre* by excess of CO_2 , fever, and toxins, either bacterial in origin, or resulting from ineffective excretion as in Bright's disease, or from defective metabolism as in diabetes, in which the diacetic acid in the blood probably acts in this way.

(11) *Hysteria*, which induces an uncomplicated tachypnœa, often extreme, but unaccompanied by signs of deficient aeration.

2. Diminished frequency of respiration

(*brachypnœa*).—Less common than tachypnœa, it may occur in cases of obstruction of the upper air-passages, shallow rapid breathing being replaced by breathing which is slow, laboured, noisy, and accompanied by stridor. In shock, hæmorrhage, and syncope the breathing may be slow and sighing, as well as in hysteria and emotional states. In conditions of increased intracranial pressure—as in depressed fracture, compression, cerebral tumours, and meningitis—the breathing is often slow as well as deep and irregular, and the same is true of the failing respiration of dying patients. Opium, chloral, chloroform, and other narcotic drugs produce a similar effect.

3. *Increased force of respiration (hyperpnœa)*.—Deep breathing is more often associated with brachypnœa, and is a common sign in intracranial disease. It may, however, accompany rapid breathing, the executive mechanism of respiration being thus directed towards promoting the greatest possible respiratory exchange. This phenomenon, known as "air-hunger," is commonly seen in diabetes, delayed anæsthetic poisoning, cyclical vomiting, uræmia, and sometimes in pneumonia, particularly in children. Respiration may be both forceful and rapid in heart disease.

4. *Shallow breathing*.—This is generally associated with rapid breathing. It is often noticeable in sleeping infants. It is the natural result of painful respiration as in pleurisy and pneumonia, and is not uncommonly seen in certain phases of meningitis.

5. *Altered respiratory rhythm*. (1) *Inspiratory dyspnœa*.—In certain cases the difficulty in breathing is confined to inspiration. This is especially so in obstruction of the upper air-passages, which furnishes the picture of dyspnœa in its purest form. The breathing may be rapid or shallow, or slow and deep, and is often accompanied by stridor. With each inspiration there is recession of the supracleavicular fossæ, the epigastrium, and the lower intercostal spaces. Descent of the diaphragm is incomplete. The dyspnœa is most urgent when its onset is produced suddenly as by a foreign body, œdema of the larynx, displacement of diphtheritic membrane, or spasm in laryngismus stridulus and the laryngeal crises of tabes.

(2) *Expiratory dyspnœa* occurs particularly in asthma, bronchitis, and emphysema, and is produced by want of elasticity of the lung and of the thoracic wall. In asthma, where the respiratory difficulty is enhanced by

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obstruction in the air-passages, the dyspnœa is urgent and distressing. This variety of dyspnœa is known by the position of the chest, which is fixed in a state of maximum expansion; the intercostal spaces and supra-clavicular fossæ are bulging, in contradistinction to the recession in inspiratory dyspnœa.

(3) *Irregular breathing* (see CHEYNE-STOKES RESPIRATION).

As well as the altered respirations, there are other symptoms which are inseparable from those of dyspnœa. *Cyanosis* is often present and is a prominent feature of urgent cases; when aeration is very imperfect it may be accompanied by jactitation. *Stridor*, essentially a sign of obstruction to the upper air-passage, may be inspiratory only, or both inspiratory and expiratory. *Stertor* accompanies dyspnœa in states of unconsciousness. In sleep it is not uncommon when tonsils and adenoids or other causes of mouth-breathing

are present, and it may be an accompaniment of old age. It is also a noticeable feature of the disordered breathing met with in coma, whether due to cerebral hæmorrhage or other intracranial lesions, or to uræmia or other forms of toxæmia. The *attitude* of a conscious patient is an index of the degree of respiratory distress. It may cause him to assume the sitting posture (*orthopnœa*) and, if intense, to seize with outstretched arms some immovable object, to serve as a *point d'appui* by the intermediation of which his accessory muscles may be brought into action. With dyspnœa of this severity the upper eyelids may be retracted, showing a rim of sclerotic and giving the patient a frightened and anxious appearance.

FREDERICK LANGMEAD.

DYSTROPHIA ADIPOSA GENITALIS
(see PITUITARY GLAND, AFFECTIONS OF).

DYSTROPHIA, MUSCULAR (see MYOPATHY).

EAR, DISEASES OF (see OTITIS EXTERNA; OTITIS MEDIA; MENIÈRE'S DISEASE; LABYRINTH, AFFECTIONS OF).

EAR, EXAMINATION OF.—In addition to such obvious signs of ear disease as deafness, tinnitus, and discharge, it should be remembered that most cases of definite vertigo, especially if prolonged beyond a moment or two, are due to aural disturbance. Further, the existence not only of vertigo but also of headache, of pyrexia, of general malaise, in suppuration of the ear, should lead to a careful investigation of that organ.

1. **Physical examination.**—This is carried out by means of reflected artificial light. An electric incandescent bulb of ground glass, provided with a bull's-eye lens, is perhaps the handiest illuminant for general purposes. For emergency work, an ordinary incandescent gas-burner, an acetylene lamp from a motor-car, or even a bright paraffin lamp, will serve the purpose perfectly, provided that the surgeon is an adept in the use of his forehead mirror.

The most comfortable forehead mirrors are

those in which the central orifice is large. Much practice is needed before one becomes habituated to the use of the mirror. (Fig. 23.)

The handiest ear speculums are Urban Pritchard's (Fig. 24), which have a circular orifice and widely expanded bell, and are easily manipulated. The practitioner requires at least three different sizes. An emergency speculum may be made by twisting into a cone a quarter of a sheet of notepaper, and cutting the apex off with scissors.

We first of all examine (under good illumination) the *external ear* and the *mastoid process*. In otorrhœa, examine the mastoid process for swelling, œdema, and tenderness on pressure. In interrogating for tenderness, apply pressure in such a way as not to disturb the auricle. Moreover, the mastoid process on both sides should be palpated, as, even when healthy, firm pressure in this region often causes pain.

Secondly, the auricle is examined with regard to its position, remembering that it projects unduly from the head in mastoid and temporal abscess, and also, though to a less degree, in the severer forms of furuncle. (See OTITIS EXTERNA).

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Attention should be paid to the presence of eczema of the auricle and meatus, especially when it complicates otorrhœa.

The *external meatal orifice* is next inspected, still without a speculum. It is here that furuncle forms, the earliest evidence of which appears in exquisitely tender spots in the meatal wall.

The speculum is now passed into the meatus in order to expose the deeper meatus and the

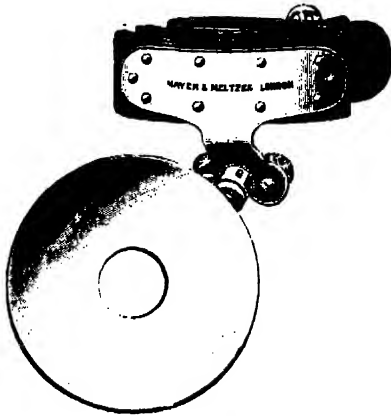


Fig. 23.—Forehead mirror.

tympanic membrane, and should be the largest that the meatus will comfortably accommodate. As the deep meatus is very sensitive, all manipulations here require a delicate touch.

The contents of the canal are first observed. In most cases epithelial debris and scales or ceruminous masses conceal the membrane. These having been cleared out of the way with an ear spud or a blunt director, look for malformations, such as stenosis or occlusion; for eczema or dermatitis of the meatal wall, for impacted cerumen, and, lastly, for discharges. Before wiping away a discharge, which may be done with a fine probe or forceps armed with cotton-wool, observe its colour, quantity, and adhesiveness. Note whether it is serous, sanguineous, or purulent.

The meatus being sharply bent at about its middle (the isthmus), we must straighten it by obliterating the angle before the tympanic membrane can be seen. If the patient is an infant of less than 2 years, pull the auricle downwards and backwards as the speculum is being inserted; if a child between 2 and 5 years, pull the auricle directly backwards; in older children and in adults, pull it upwards and backwards.

In infants the external meatus is extremely short, and the membrane is situated very obliquely, facing almost directly downwards. It there is any difficulty in making a satisfactory examination of the ear in a child, a general anæsthetic should be administered.

Narrowing or stenosis of the meatus may be due to furuncle (common), to exostosis (rare), to eczematous infiltration, or to bulging of the roof or postero-superior wall from suppuration in the mastoid antrum. In the last instance the bulging is situated deeply, near the membrane. In furunculosis, on the other hand, the seat of the swelling is in the outer section of the meatus.

The normal *membrana tympani* is pearly-grey or white, semi-translucent, and polished. It is seen obliquely, as it faces downwards and forwards as well as outwards.

The landmarks of the membrane are—(1) the handle of the malleus, the most prominent feature, which is inclined downwards and backwards to its (2) tip or umbo, lying a little below the centre point of the membrane, with (3) a triangular reflex of light passing from the umbo downwards and forwards to the periphery of the membrane. At the upper end of the handle of the malleus is a small projection, (4) the short process of the malleus. Arching forwards and backwards from the short process are (5) the two curved lines

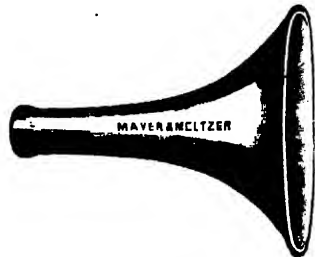


Fig. 24.—Urban Pritchard's ear speculum.

which separate the vibrating membrane below from the *membrana flaccida* or Shrapnell's membrane above. (Fig. 25.)

The colour, thickness, and transparency of the membrane should be observed, and atrophic areas, scars, depressions, perforations and (in suppuration) granulations, and polypi should be looked for.

The membrane may be retracted (indrawn), or it may bulge in whole or in part. The recognition of a retracted membrane is of some importance, as it is often due to curable

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Eustachian obstruction. The following are its distinctive features: (1) The handle of the malleus, having an excessive inward tilt, appears shorter than usual (from the foreshortening); (2) the short process of the malleus juts out very prominently; (3) the curved lines separ-

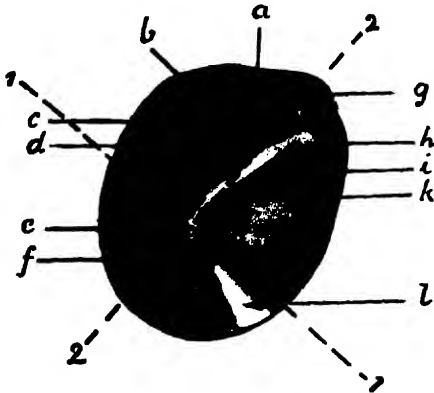


Fig. 25.—The drum membrane, enlarged. (Porter's "Diseases of the Throat, Nose, and Ear.")

a, Posterior fold; b, long process of incus; c, tendon of stapedius muscle; d, head of stapes; e, umbo; f, shadow of niche to fenestra rotunda; g, Shrapnell's membrane (membrana flaccida); h, short process of malleus (processus brevis); i, anterior fold; k, handle of malleus (manubrium mallei); l, light reflex. 1, 1, and 2, 2, Imaginary lines dividing the drum membrane into four quadrants.

ating the membrana vibrans from Shrapnell's membrane are abnormally distinct. In chronic cases the tip of the malleus handle and the membrane around it may become adherent to the promontory on the inner wall of the tympanum.

A "bulging" membrane is due to the accumulation of fluid in the middle ear.

A useful modification of the ordinary ear speculum exists in Siegle's pneumatic speculum (Fig. 26), by means of which the mobility of the membrane and of the handle of the malleus can be tested. With regard to mobility, note whether the membrane moves freely in and out, and whether the handle of the malleus accompanies the membrane in its movements, or whether there seem to be any adhesions limiting their excursions.

The beginner should not despair if he sometimes finds it difficult to be sure of what he sees in the ear. This experience occasionally afflicts even the expert.

Another method of investigating the condi-

tion of the ear is to listen to the sounds produced when the tympanic cavity is inflated through the Eustachian tube by means of Politzer's bag or the Eustachian catheter. In this way we receive information regarding (1) the patency of the Eustachian tube; (2) the contents of the tympanum; (3) the presence or absence of perforations in the membrane.

Politzer's method.—An auscultation tube connecting the patient's ear with the surgeon's ear is inserted. The nozzle of the Politzer bag is put into the patient's nostril, and his nose is closed against it with the fingers. Then, while the patient, with closed lips, forcibly blows out his cheeks, the bag is suddenly but gently compressed, and, if the Eustachian tube is patent, a faint click or snap is distinctly audible as the membrane is forced outwards by the rising air-pressure within the tympanum. (When Politzer's bag is used as a therapeutic measure the compression has to be much more forcible.)

Politzer's method is most useful in diagnosing perforations in the membrane: If the membrane is perforated, inflation by Politzer's method induces the "perforation sound," a whistling or blowing produced by the escape of air through the perforation.

The Eustachian catheter.—The auscultation



Fig. 26.—Peter's modification of Siegle's pneumatic speculum.

tube being in position, pass the catheter point downwards through the corresponding nostril until it is felt to impinge upon the posterior wall of the pharynx, then bring it forward about $\frac{1}{4}$ in., rotating the instrument until its point is directed upwards and slightly outwards. This brings it to the orifice of the Eustachian tube. Then inflate with the bag.

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When the Eustachian tube is patent, the air rushing through it into the tympanum sets up a characteristic blowing sound audible by the surgeon through the auscultation tube. When the tube is blocked by swelling, the sound is faint and distant, while the presence of fluid in the tympanum is betrayed by moist râles or bubbling. The perforation sound heard through the catheter is peculiarly characteristic.

It must not be forgotten that as part of the examination of the ear a careful examination of the nose, naso-pharynx, and pharynx is necessary, especially for nasal catarrh or suppuration, and for adenoids.

2. Functional examination.—The tests employed in the examination of hearing have a double object: (1) they seek to estimate the amount of hearing present; (2) they inform us whether it is the conducting apparatus that is chiefly or solely at fault. Deafness due to interference with the conducting apparatus, known as "obstructive deafness," presents certain characteristics which distinguish it from deafness due to interference with the perceptive apparatus, which is known as "perceptive deafness" or "nerve-deafness."

We are able to determine, from the findings obtained on testing the hearing of a deaf person, whether the lesion responsible for his defect is situated in the conducting apparatus or in the perceptive apparatus, or in both.

When the hearing tests manifest the characteristics of obstructive deafness, we know that the lesion must lie in the external auditory meatus, the tympanic membrane, the chain of ossicles, the tympanic cavity, or in the Eustachian tube. When they show the deafness to be nerve-deafness, we know that the lesion must be one affecting the cochlea, the cochlear branch of the auditory nerve, or its tracts or centre in the brain.

Hearing-tests: conversational voice.—The *method* is as follows: The patient stands sideways to the examiner with the ear to be tested towards him, the distal ear being stopped with the finger. The examiner withdraws to a distance of about 24 ft., and utters the test-word or sentence, the patient being asked to repeat what he hears. If the word is not correctly repeated at 24 ft., the examiner takes a step nearer, and again utters the word; and so on until the patient succeeds in repeating correctly what has been said. The distance intervening between examiner and the patient is then noted.

Richly vowelled words like "America,"

"tomato," "potato," and numbers like "four-hundred-and-fifty-two," and so on, are the most suitable for the purpose. Those with sibilants should be avoided.

If the conversational voice can be clearly heard at about 20–30 ft., the hearing for the voice may be written down as normal. Any distance less than that indicates defect.

Whisper.—The whisper, being more uniform, is generally regarded as a more reliable test than the speaking voice. We employ what is termed the "unforced whisper," which carries a considerable distance. For ordinary purposes, however, the range of its audibility may be taken as extending to 24 ft. or more. The *method* adopted is the same as that for the voice, the only difference being that the test-word or sentence is whispered instead of being spoken aloud.

Acoumeter or watch-tick.—The acoumeter is a simple instrument constructed to emit a clicking or tapping sound. Scientifically it is superior to a watch, as all acoumeters of the same pattern produce a sound of the same character. Yet it is the watch which is generally employed, and if the normal distance be known at which the tick of any particular watch is heard, the test is fairly accurate.

Method: Hold the watch beyond the farthest point at which it can be heard normally, and approach it gradually to the patient's ear. His eyes should be closed. Get him to indicate when first he hears the tick, and note the distance in inches. The finding may be written as a fraction: $\frac{a}{b}$, for example, means that the patient cannot hear at more than 6 in. distance a watch which he ought to be able to hear at 32 in.

Tuning-fork tests.—The tuning-fork used for ordinary testing is that corresponding to middle C (256 V.D.), provided with a flattened disc-like end to facilitate its close application to the mastoid region. It emits a pure musical tone, and is used to test the patient's hearing for sounds transmitted both through the air and external meatus, and through the bones of the head, as it is by a comparison of these two routes of conduction that the distinction is drawn between obstructive deafness and nerve-deafness.

Test for hearing by air-conduction (through the external meatus).—Sound the fork by striking it upon the knee. While it is vibrating hold it close to the external meatus of the ear to be tested, in such a position that the two prongs are in line with the meatus. When the

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patient ceases to hear the sound, transfer the fork to your own meatus and note by how many seconds you can hear it longer. Express the difference in seconds preceded by a minus sign, thus: Meatus = -15 secs.

Test for hearing by bone-conduction.—Bring the disc-shaped end of the vibrating tuning-fork into close contact with the mastoid of the ear being examined. When the patient ceases to hear it, transfer it to your own mastoid, and note whether or not you hear it. If you do so, then the patient's bone-conduction is diminished. Note, as in testing the air-conduction, the difference in seconds, and express the finding thus: Mastoid = -8 secs.

If, on the other hand, you cannot hear the sound when the patient ceases to hear it, strike the fork again, hold it on your own mastoid, and when the sound stops apply it to the patient's mastoid. If he is now able to hear it, his bone-conduction is increased: the finding is written in seconds with a plus sign.

A decreased bone-conduction (*minus* mastoid) denotes nerve-deafness, whilst an increased bone-conduction (*plus* mastoid) indicates obstructive deafness.

The next two tests (Rinné's and Weber's) are useful in emphasizing the difference between the air-conduction and the bone-conduction.

Rinné's test is the more important. By it we compare the air-conduction with the bone-conduction of the same ear.

Method.—Apply the vibrating fork to the patient's mastoid, and when he ceases to hear it, transfer it to the meatus of the same ear without again striking it. If he can hear it there, the Rinné is *positive*, the air-conduction of that ear exceeding the bone-conduction.

If there is decided deafness, this will mean that the bone-conduction is reduced, and that the patient is suffering from nerve-deafness. It should be noted, however, that Rinné's test is positive in health.

In obstructive deafness the bone-conduction exceeds the air-conduction, so that when the sound of the fork at the meatus has died away the patient can still hear it for a few seconds through the bone. This is termed a "Rinné negative."

The **vertex or Weber's test** compares the bone-conduction of the one ear with that of the other ear. Sound the fork, place the disc in firm contact with the vertex of the patient's head, and ask him in which ear, if either, he hears it the louder or the longer. If the hear-

ing power in the two ears is decidedly unequal, the sound will be lateralized to the deaf ear in obstructive deafness, whilst in nerve-deafness the sound will be lateralized to the better ear.

This test often proves to be unreliable, depending as it does upon the ability of the patient to analyse his own sensations accurately. Many patients with undoubted obstructive deafness in one ear believe that they hear the fork better in the sound ear because they expect to do so.

In the tuning-fork tests the ear which is not being tested must not be stopped with the finger.

Estimation of the upper and the lower limit of hearing.—In health the upper tone limit reaches as high as 40,000 V.D. per second in childhood and adult life. After 60, however, the upper limit of tone perception is lowered, a fact which must be allowed for in testing.

High tones.—The Edelmänn-Galton whistle produces a shrill note which can be raised or lowered in pitch as desired. Markings upon the stem of the whistle correspond approximately to the number of vibrations produced at the different levels. A decided lowering of the upper tone limit, as estimated by the Edelmänn-Galton whistle, in combination with a diminished bone-conduction for the tuning-fork, indicates nerve-deafness.

Low tones.—Loss of hearing for the deep tones is caused by obstructive deafness. For this part of the examination the deep tuning-forks (from 64 to 128 V.D.) are employed. In testing, the air-conduction only is interrogated.

Synopsis of findings.—First we estimate the *amount of deafness* present by (1) the farthest distance at which the patient can hear the voice, the whisper, and the watch-tick; and (2) by the amount of reduction for tuning-forks of a middle tone as compared with the normal.

Secondly, with regard to the *type of deafness* present, we consider the deafness to be *obstructive* if—

- (1) The bone-conduction exceeds the normal (*plus* mastoid).
- (2) The bone-conduction is increased relatively to the air-conduction (*minus* Rinné).
- (3) The hearing for low tones is abolished or reduced.
- (4) With the tuning-fork on the vertex of the head the sound is lateralized to the deaf ear.

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On the other hand, we consider the deafness to be *perceptive nerve-deafness* if—

- (1) The bone-conduction is less than the normal (*minus* mastoid).
- (2) The bone-conduction is decreased relatively to the air-conduction (*plus* Rinne).
- (3) The upper tone limit is lowered, while the low tones are audible.
- (4) With the tuning-fork on the vertex the sound is lateralized to the deafer ear.

Mixed obstructive and nerve-deafness.—It not infrequently happens in severe cases of deafness that the bone-conduction is less than normal, whilst Rinne's test indicates obstructive deafness. In such cases both nerve-deafness and obstructive deafness are present, the lesion affecting both the conducting and the perceptive apparatus. These, as a rule, are cases in which severe obstructive deafness has been followed by atrophic changes in the labyrinth.

We must not rest our diagnosis, especially when the deafness is slight, solely upon the data obtained from the hearing-tests. All the circumstances of the case—the findings obtained from the physical examination of the ear and of the patient, and the effect upon the deafness produced by inflation with Politzer's bag and with the Eustachian catheter—should be taken into account before forming an opinion upon the probable seat of the lesion which has produced the deafness in any particular case.

Vestibular system (semicircular canals; canicular system).—We investigate the condition of the vestibular system with the idea of diagnosing the extent of disease in the labyrinth, especially in suspected purulent labyrinthitis. The state of the vestibular reflexes often facilitates also the diagnosis of intracranial disease, whether secondary to ear mischief or not. Therefore the vestibular tests should be taken in all cases of nerve-deafness, and also in all cases of suspected disease of the brain or spinal cord.

Cochlear lesions are often accompanied or followed by changes in the semicircular canals. Probably, also, the converse is true, speaking broadly. But, at the beginning, either system may undoubtedly be affected independently of the other.

If the stimuli reaching the nerve-centres from the semicircular canals of one side preponderate over those from the canals of the other side, a sense of loss of equilibrium—

vertigo or giddiness—is produced. Along with this vertigo there appears a deviation of the muscles of the body, including those of the eyes. In the eyes the deviation is converted into a peculiar kind of nystagmus, known as *vestibular nystagmus*. It consists of two movements, first a slow deviation of the eyeballs in one direction, followed by a rapid twitch of the eyeballs back again to their original position. These unequal oscillations follow each other rapidly, and, once seen, are unmistakable. A further peculiarity of vestibular nystagmus is that the nystagmus is exaggerated when the person turns his eyes in the direction of the rapid twitch, and is diminished or abolished when he turns his eyes in the direction of the slow deviation. For this reason we speak of the nystagmus as being *directed* to the side of the rapid twitch. Thus, nystagmus "to the right" means that the rapid twitch is directed to the right, and that the nystagmus is increased on turning the eyes to the right, and vice versa.

This sign occurs in certain diseases of the labyrinth. If, for example, a sudden pathological blow, such as hæmorrhage or acute suppuration, destroys the vestibular end-organs on one side and paralyses their function, then we find *spontaneous* vestibular nystagmus when we examine the eyes. (See LABYRINTH, AFFECTIONS OF.)

Nystagmus and vertigo may also be elicited by excessive *artificial* stimulation of healthy semicircular canals by (1) rotation of the body (as children do in play); (2) heating or cooling the external auditory meatus by hot and cold water; and (3) galvanism.

It is the first two methods which we habitually employ in our clinical tests of the activity of the vestibular system.

Vestibular reaction tests. (1) *Rotation method.*—The patient is seated, with his feet clear of the ground, on a rotating chair or stool. His eyes should be covered with shaded spectacles. He is then rotated with the head erect in one direction at the rate of ten revolutions in twenty seconds. After ten revolutions the rotation is suddenly stopped, and the eyes at once examined. If the vestibular system is normal, there will be well-marked nystagmus affecting both eyes with its direction (i.e. the direction of the short twitch) *towards the side from which the patient was rotated.*

The nystagmus lasts about thirty seconds, and subsides gradually. Definite but not excessive vertigo is experienced for a few moments

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after the cessation of the rotation. It passes away before the nystagmus disappears.

When we wish to ascertain the condition of the semicircular canals on the right side, we rotate the patient from right to left. On the other hand, when we wish to test the canals on the left side, we rotate the patient from left to right, and look for nystagmus to the left.

We come now to discuss the effects of rotation (a) when the vestibular system of one side is inert; (b) when the vestibular system on both sides is inert.

(a) When the vestibular system of *one side* is inert, the other being active, rotation from the diseased side will be followed by a feeble response, while rotation from the healthy side will be followed by a normal response. For example, if the right labyrinth is destroyed or paralysed, rotation from right to left will have a feeble result, the nystagmus lasting only about 10 sec., and there being little or no vertigo. On the other hand, if we rotate the patient from left to right, the left labyrinth will be chiefly stimulated, and, if it is healthy, a normal nystagmus lasting 30 sec., and accompanied by vertigo, will be produced.

(b) When the vestibular system of *both sides* is destroyed, rotation to either side will produce little or no nystagmus, and no vertigo.

(2) *Caloric nystagmus*.—Raising or lowering the local temperature of the external auditory meatus induces nystagmus and vertigo in health. When cold is employed the nystagmus is directed to the opposite side of the ear tested; when heat is employed the nystagmus is directed to the same side. Cold, in the form of cold water, gently injected, or run from a small douche can, into the meatus, is what is generally employed.

The great advantages of this test are that it is easily applied, and that it interrogates definitely only one ear at a time.

Method.—After making sure that the meatus is not blocked with cerumen, granulations, or polypi, water at a temperature of about 68° F. is slowly injected into the meatus of one ear, the patient being directed to turn his eyes meanwhile to the opposite side. At the first appearance of nystagmus the injection is stopped. In health, nystagmus will appear in about 30 sec., and is accompanied with mild vertigo. If the vestibular system is inert—as in suppuration of the labyrinth—little or no nystagmus appears even after prolonged syringing. If, on the other hand, it is irrit-

able, as in neurasthenic conditions, the reaction appears early, and is severe, being accompanied with great vertigo, nausea, and sometimes vomiting. Should such an excessive reaction set in, the unpleasant symptoms may be at once checked by the injection of warm water into the meatus.

After one ear has been tested, the other is likewise dealt with, but an interval of at least 15 min. should elapse between the two tests.

When the membrana tympani is perforated the caloric reaction appears quickly, because the cold water comes into direct contact with the wall of the labyrinth.

Synopsis of results obtainable from the vestibular tests.—Both vestibular systems are healthy if—

- (1) Rotation to either side is followed by well-marked nystagmus directed towards the side from which the patient was rotated, accompanied by definite, but not severe, vertigo, and lasting about 30 sec.
- (2) Cold water in either meatus produces well-marked nystagmus to the opposite side, accompanied by moderate vertigo (with water at a temperature of 68° F. the reaction appears in about 30 sec.).
- (3) Hot water in either meatus induces similar results, but with the nystagmus directed to the same side.

Both vestibular systems are totally inert if—

- (1) Rotation to either side is followed by no nystagmus and no vertigo.
- (2) Neither cold nor hot water in either meatus induces nystagmus or vertigo.

The vestibular system of *one side is inert and the other healthy* if (assuming the right side to be inert and the left to be active)—

- (1) Rotation from right to left is followed by feeble nystagmus lasting only about 10 sec., and not accompanied by vertigo.
- (2) Rotation from left to right is followed by well-marked nystagmus lasting about 30 sec., along with moderate vertigo.
- (3) Cold water in the right external meatus does not induce nystagmus and vertigo.
- (4) Cold water in the left external meatus does induce definite nystagmus and vertigo.
- (5) The same holds good of hot water.

When the reactions are delayed, shortened,

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and imperfect, although still present, a partial destruction of the vestibular system may be diagnosed. This is especially common in old-standing labyrinth lesions affecting hearing in the first place. In functional and hysterical deafness, on the other hand, the vestibular reactions are, as a rule, normal.

DAN M'KENZIE.

EAR, FOREIGN BODIES IN (*see FOREIGN BODIES IN THE EAR*).

ECHINOCOCCUS (*see HYDATID DISEASE*).

ECLAMPSIA, INFANTILE (*see CONVULSIONS*).

ECLAMPSIA, PUERPERAL (*see PUERPERAL ECLAMPSIA*).

EOTHYMA (*see IMPETIGO CONTAGIOSA*).

ECTOPIQ GESTATION (*see PREGNANCY, EXTRA-UTERINE*).

ECTROPION (*see EYELIDS, AFFECTIONS OF*).

ECZEMA (including Dermatitis from External Irritants).—Eczema is not a disease of well-defined etiology. It is the name given to a superficial inflammation of the skin in which the papillary layer and epidermis are chiefly involved; in which serous effusion, with the formation of microscopic or macroscopic vesicles in the epidermis is a marked feature; and in which there is an irregular formation of the horny layer, called parakeratosis. The lesions occur in patches, are very irritable, and tend to recur. Eczema is, therefore, a catarrh of the skin, comparable to that occurring in the mucous membranes.

The various lesions of eczema can be produced by the application of certain irritants to the skin; it is not certain, however, whether similar lesions can be produced by toxins circulating in the blood. Cases in which we know the offending agent we label "dermatitis," prefixed by the name of the substance producing the inflammation (e.g. formalin dermatitis), while we reserve the term eczema for those cases in which the etiological factor is unknown.

We shall deal first with the forms of superficial dermatitis produced by external irritants, sometimes called *dermatitis venenata*, and then with what, by way of distinction, may be termed eczema proper.

DERMATITIS FROM EXTERNAL IRRITANTS

Etiology.—The skin is exposed to irritants of many kinds, which, for simplicity, may be

divided into five groups: (1) Mechanical, (2) thermal, (3) actinic, (4) chemical, and (5) bacterial. With the last group we shall not deal in this article, as it is considered elsewhere under appropriate headings.

The lesions produced by any of these irritants vary not only with the intensity of the irritant and the time that it is applied, but also with the susceptibility of the patient. To illustrate this last point, it is common knowledge that in a group of french-polishers only one or two will develop a trade dermatitis while the rest remain immune, although equally exposed to the irritant concerned.

The dermatitis from an irritant is usually limited to the area to which the irritant is applied, and the inflammation subsides as soon as the irritant is removed. This is not always borne out clinically, because before the original irritant has been removed another may have appeared on the scene. Thus, in a case of moist dermatitis produced by an external irritant the discharge may act as an irritant and spread the inflammation beyond the limits to which the original irritant was applied.

Pathology.—The effect of the application of an irritant is to produce a reaction of the skin in an effort to rid itself of the irritant, so that all that is visible to the naked eye, in the milder forms, is this reaction. The direct action of an irritant is only seen when the action is so violent or prolonged that actual destruction of portions of the skin occurs. Reaction in its mildest form consists of a dilatation of the papillary vessels, which produces erythema, followed by some oedema of the papillary layer and of the mucous cells of the epidermis, together with a cellular exudate into the papillary body. This leads in the mildest cases to an imperfect cornification, so that scaling occurs, or if the oedema is more pronounced small vesicles appear under the horny layer. When a more powerful irritant is applied the congestion and oedema are greater; the area affected becomes intensely red, hot, and swollen, and larger vesicles or bullæ tend to form on the surface.

The contents of the vesicles and bullæ thus formed are found to be free from organisms if cultures are taken early, but after a short time, if the lesions are unruptured, staphylococci may be found.

Symptomatology.—Irritants externally applied may produce, apparently, a dermatitis in at least three different ways:

1. If a fairly intense irritant is applied, for

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instance a mustard plaster, a uniform dilatation of the papillary vessels takes place with an exudation of fluid into the epidermis and papillary layer, followed often by a cellular exudation. As a result, this area becomes intensely red, and in the more severe cases pin-head vesicles or even large bullæ appear on the surface; these may rupture and give rise to "weeping." The fluid which escapes is highly albuminous, and stiffens linen, or, if it is allowed to dry on the skin, forms thin transparent crusts.

2. If a milder irritant is rubbed over the skin some of it will tend to lodge in the lanugo hair-follicles, and as these are the weakest points in the protective apparatus the lesions will start there. In this case pinhead-sized red papules, usually surmounted by a minute vesicle, appear at the follicular openings; they tend to aggregate into patches consisting of an erythematous base with numerous papules or papulo-vesicles scattered through it. The patch may remain dry and scale freely, or may become converted into a weeping dermatitis.

3. This group appears to arise mainly from the effects of damage to the horny layer by drying. In people who have their hands continually in water, and especially if alkalis like soap and soda are added, there is a tendency for the horny cells to soften and take up water. If these are rapidly dried, especially by a cold wind or a hot fire, the cells tend to break away from their neighbours and expose the sensitive mucous layer; this is particularly liable to occur in the furrows of the skin. A subacute dermatitis is thus set up which may undergo the secondary changes mentioned above.

These three processes give rise to what are usually called the primary lesions of eczema. The first produces the erythematous type, the second the papular and papulo-vesicular; the third, though primarily of the chronic erythematous type, is so persistently associated with scaling that it may almost be classed as a primary squamous type. A primary pustular type is commonly described, but though follicular pustules may develop from a non-bacterial irritant, such, for instance, as a 1-in-1,000 perchloride-of-mercury dressing, they are usually quite evanescent, while the pustules that persist are of definite bacterial origin; it is better, therefore, to consider these cases as pyodermatoses.

All these primary types may undergo secondary changes. Thus, scaling may occur (*eczema squamosum*) from improper formation of the

horny layer (*parakeratosis*), or weeping (*eczema rubrum*) from rupture of the vesicles. Crusting (*eczema crustosum*) is produced by drying of the exudate. Chronic oedema and cellular infiltration produce a thickening and rigidity of the skin with a tendency to produce cracking in the deeper furrows, especially on the palms and soles (*eczema rimosum*), or may produce a special form of thickening called *lichenification*, to be described later. In extreme cases, as a result of associated lymphatic obstruction, a warty overgrowth of the epidermis (*eczema verrucosum*), or even an elephantiasis, may occur. Secondary infection with pyogenic organisms is an almost invariable accompaniment of the moist forms, and here the crusting becomes much more pronounced (*eczema impetiginosum*).

1. **Mechanical irritants.**—The form of irritation which produces the most varied types of eruption is scratching and rubbing. Scratching causes erection of the hair-follicles, and as these offer an obstruction to the finger-nail their tops may be scratched off and a bloodstained crust form at these points; further than this, linear scratches may be seen. In the more severe forms "eczematization" takes place, that is to say, numerous closely placed papules or papulo-vesicles form on a red base, the whole presenting a circumscribed patch either covered with thin scales or moist and oozing. If the scratching is still continued the affected skin becomes thick and dry, the surface has a tessellated appearance, due to the abnormal depth of the lines of the skin, and the red colour is replaced by a dark purple; this stage is spoken of as lichenification, which is usually seen in the flexures of the knees and elbows, around the ankles or nape of the neck, but may develop at any site of irritation; it is, however, generally localized. Eczematization may often be generalized—for instance, in the pruritus caused by pediculosis. The most severe form of skin damage resulting from scratching is the linear ulcer, seen chiefly on the lower extremities in those suffering from pediculosis vestimentorum, and very common during the War.

2. **Thermal irritants.**—The effects of heat are seen in burns and scalds. In both these conditions an intensive erythema is produced, which in the more severe cases is associated with blister-formation. The later stages are considered in the article on BURNS AND SCALDS. Frequent exposure to less intense heat gives rise to a persistent erythema, followed by pig-

mentation, which often forms a curious wide-meshed reticular pattern. This is seen chiefly about the front of the shins in certain trades, or in old people who sit all day long in front of the fire, and is termed *ephelis* (or *erythema*) *ab igne*. Severe cold, beyond producing a transitory hyperæmia, causes little or no superficial dermatitis, the changes being mainly of a vasomotor disturbance.

3. **Actinic irritants.**—Light, whether the direct rays of the sun or from artificial sources, produces marked inflammatory changes in the skin. The direct sun's rays produce the well-known *erythema solare*, which, when it subsides, leaves behind pronounced pigmentation of the exposed areas. In the more severe cases, and especially in certain susceptible individuals, blistering occurs. Exposure to the sun for prolonged periods may lead to degenerative skin changes, with atrophy, pigmentation, telangiectases, and warty formations, the last of which may become epitheliomatous. This type of skin is seen in white people who have spent years in the tropics, and is often spoken of as "tropical skin." It does not appear to occur in the dark races, presumably owing to the protective action of their pigment against the sun's rays. Much more severe dermatitis is produced by X-rays and radium (see DERMATITIS, X-RAY).

4. **Chemical irritants.**—These are responsible for by far the largest and most important group of superficial dermatoses, a group which probably includes a large number of cases in which the irritant cannot be found and which, for the sake of convenience, we classify under the term eczema.

The very numerous chemical agents capable of producing a dermatitis may roughly be divided into five classes: (1) Animal irritants, (2) vegetable irritants, (3) medicinal agents, (4) substances used in trades, (5) decomposed body secretions.

(1) **Animal irritants.**—The bites and stings of insects scarcely come into our group of "eczematous" dermatoses, and the only insects which produce this type of lesion are certain caterpillars, mainly of the hairy type, such as the "woolly bear" in this country and the caterpillar of the "brown-tail moth" in certain parts of America. Some people are very susceptible to these insects, and the lesions are probably produced by the hairs, which bear an irritant that penetrates the skin; in fact, the mechanism is the same as in the stinging-nettle. In some cases an urticarial reaction is

produced, in others an acute erythematous dermatitis.

(2) **Vegetable irritants.**—Plants act as irritants in two ways, either by perforating the skin with their fine bristles, e.g. the stinging-nettle, or by the contact of their juices, which contain an irritating oily substance that dissolves in the sweat. In the former case there is usually an urticarial reaction, nettle-rash, which is familiar to everyone; in the latter an erythematous dermatitis of varying intensity is caused. The plants which produce the most violent dermatitis are those of the rhus (or poison ivy) group, which are not met with in Britain. The action of this poison on the skin is closely comparable to that produced by mustard gas; some persons, however, have a distinct predisposition to be attacked. The parts chiefly affected are the uncovered parts of the body—the face, hands, and forearms—but in the more severe cases the moist parts of the body are attacked, even when protected by clothing; thus the genitals, axillæ, and flexures, and even the whole body may be affected. In mild cases a simple erythema is produced, associated with a good deal of itching, and followed by desquamation and transitory pigmentation. In the more severe cases an intense erythema occurs associated with œdema of the subcutaneous tissues, so that the face, and especially the eyelids, become much swollen. Vesicles and bullæ develop on the affected areas, and these may rupture and produce a weeping or crusted dermatitis. Rarely, necrosis has resulted. In these cases the patient complains of severe burning of the skin, followed later by itching.

The plant that most often causes dermatitis in these islands is the *Primula obconica*; but other varieties of the primrose, and other plants, among them *Daphne mezereum*, oleander, rue, parsnip, daffodil, and chrysanthemum, may act in the same way. The dermatitis is the same as in the case of the rhus group, but usually less severe, being generally confined to the exposed parts and taking the form of an erythematous or vesicular dermatitis. Handling plants is not always necessary to the production of a dermatitis, for very susceptible persons may be affected in this way simply by entering a room containing a primula. The handling of certain woods, such as teak, satinwood, and ebony, may also produce a similar reaction.

(3) **Medicinal agents.**—It is not only substances used in medicine as counter-irritants,

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such as cantharides, mustard, and croton oil, that set up dermatitis, but many others also, such as sulphur, chrysarobin, mercury, and iodoform. (See DRUG ERUPTIONS.)

(4) *Chemical agents used in trades.*—The commonest of the many forms of dermatitis met with in trades is that due to the excessive use of soap, soda, and water, the so-called "washerwoman's eczema." This, as has been explained above, is probably largely due to the effects of rapid drying, rather than of the direct action of the soap and water. The lesions are more or less circumscribed patches of dermatitis, either of the dry squamous or of the papulo-vesicular type, on the backs of the hands and forearms, or they may take the form of deep cracks in the neighbourhood of the nails or a deep-seated vesicular eruption of the fingers and hands. This kind of dermatitis is most likely to occur in the winter, and clears up when the weather gets milder. Recurrences are very common, unless the condition is dealt with at once and preventive measures are taken, and each attack is usually more extensive than the previous one. Eventually the dermatitis may cease to be local and lesions may appear on other parts of the skin, as, indeed, may occur in dermatitis from any local irritant. These disseminated cases will be considered later.

Similar types of eruption are met with in grocers and bakers—it is thought from handling sugar and flour, though in bakers heat is probably an important factor; in french-polishers, photographers, dye-workers, and in many other trades. Workers engaged in handling explosives are liable to a special type of acute vesicular dermatitis of the hands closely simulating dysidrosis or cheiropompholyx. Constant contact with petrol and paraffin oil leads to a chronic follicular hyperkeratosis of the hair-follicles of the parts affected, and a similar condition may occur in tar-workers. In both these groups of patients there is a tendency to epithelial proliferation, with the formation of warty growths which may become epitheliomatous.

(5) *Decomposed body secretions.*—Decomposed sweat, urine, etc., may set up a dermatitis, usually of the erythematous type. This is seen in fat women under the breasts or in the folds of the groin, or in infants in the napkin region. An extensive erythema develops, the horny layer becomes separated, and a moist, intensely red, raw area results; this condition is commonly spoken of as "intertrigo." Friction

undoubtedly plays a part in the production of these lesions, but they do not occur unless the parts are sodden by discharges.

Dermatitis artefacta.—In connexion with dermatitis due to external irritants, it is necessary to give some account of self-inflicted eruptions. They are usually met with in hysterical persons, whose object is to induce sympathy, or in persons who are endeavouring to secure compensation or avoid some unwelcome duty. The lesions are produced by such means as friction, the application of strong acids or alkalis or of blistering fluids, or of heat, or with the aid of a sharp instrument. They may be erythematous or ulcerative; they may be single or multiple; and they are found on parts of the body easily accessible to the hands, and especially to the right hand (except in left-handed people, where the reverse is the case). Usually they are very characteristic, especially when a liquid agent has been used. They have sharp edges and the outline is angular, unlike that seen in any ordinary skin eruption, and not infrequently irregular patches near the main lesions have just the character of spilt fluid. Dry aseptic sloughs are often present. It must further be noted that the artefact may be a lesion due to some perfectly innocent cause that is being kept open.

In hysterical cases anaesthesia of the palate is often present.

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We have now to consider the group of catarrhal dermatoses which, although presenting cutaneous lesions similar to those described, cannot be traced to any definite irritant.

Etiology and pathology.—The nature of these eruptions has long been in dispute. Are they produced entirely by an external irritant which has not yet been discovered, or are they due solely to the presence of toxins of a bacterial or metabolic nature circulating in the blood-stream, or do both factors play a part in their production? We have already noted that some persons are more prone than others to a dermatitis produced by a known chemical irritant; it is not unreasonable, therefore, to assume that certain persons may react pathologically to irritants to which everyone is daily and hourly exposed, such as the air, the sun, soap and water used for ordinary washing purposes, even the friction of clothes. The probability is that most local "eczemas" are really the reaction of local areas of skin to

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extremely mild irritants in those whose skin, for some reason or other, is for the time being in an abnormally sensitive state. The possible causes of such predisposition are numerous, but nothing very definite is known about them, and it is impossible, therefore, to dogmatize. Local predisposition may be brought about by a previous dermatitis, for it is well known that an area once affected is very liable to subsequent attacks, no matter what the cause of the first attack. Abnormalities of the skin and its secretions are potent predisposing causes. Thus patients with xeroderma, in which there is a virtual absence of sweat and sebaceous secretion, are very liable to "eczematous" eruptions. Hyperidrosis, in which there is excessive sweat secretion, and seborrhœa, in which there is an excessive sebaceous and sweat secretion, may predispose to attacks of eczema. Circulatory disturbance, such as varicose veins or chronic vascular stasis, met with in the lower extremities in middle-aged and old people, are a frequent predisposing cause of the chronic eczemas of the legs. Various toxic conditions such as gout and rheumatism; digestive disturbances such as constipation and dyspepsia; deficient elimination, as in nephritis; chronic infections such as pyorrhœa and tonsillar sepsis; and alcoholism, have been considered responsible for some cases.

Disturbances of the nervous system have also been held by some to predispose to eczema; among these may be mentioned teething in infants, uterine troubles, and the neuroses produced by shock, worry, and overwork.

There is a distinct tendency for local eczemas to spread to other parts of the body; this is even observed in cases where the original dermatitis was produced by a known irritant. It is possible that in these cases we have to deal with an acquired hypersensitiveness, an anaphylactic phenomenon. It is clear that in such patients the conveyance of secretion from an eczematous patch to a healthy area of skin may determine a fresh patch of the disease, whereas such a fresh patch could not be so produced in another individual. This, of course, proves nothing in those cases in which some form of predisposition has to be invoked to account for the initial attack, but when it occurs in association with a dermatitis of known artificial origin the presence of an acquired hypersensitiveness is at least probable.

Even when possible cases of hypersensitiveness have been excluded, we are left with a certain number of cases in which it is difficult

to see how any external irritant comes into play; it is to cases of this class that a purely internal cause such as those mentioned had been assigned. Further knowledge of the chemical processes occurring in the body is necessary for the solution of this difficulty.

The anatomical changes in the skin have already been described.

Symptomatology.—It has been customary to describe eczemas as acute and chronic; but these terms are very loosely used, and in practice one meets with every degree of intensity and persistence of eczematous lesions, so that it is difficult to lay down any hard-and-fast rules for such a division. It is better, therefore, not to lay too much stress on this classification.

The lesions of eczema are similar to those described in the first section of this article. As they are so varied, they will be described as they occur in the regions most frequently affected.

Face and scalp.—In infants a vesicular or papulo-vesicular eczema of the face and scalp is common. It is generally met with between the sixth and eighteenth months, and tends to last about six months. Most commonly it begins on the *cheeks*, and spreads rapidly, involving the whole face and scalp. It is intensely irritable, and the irritation appears to come on in attacks, during which the child endeavours violently to rub or scratch itself. As a consequence of rubbing, the vesicles rupture and "weeping" ensues; secondary infection follows and, as the secretion dries, the lesions become covered with crusts. Occasionally the weeping is not a pronounced feature, and a scaly erythematous dermatitis occurs. The cause of this condition is not at all clear. It has been attributed to improper feeding, teething, etc., to seborrhœa, and also to external irritants such as soap and water used carelessly. None of these explanations covers all cases, but, whatever may be the origin, there can be little doubt that the lesions are spread by rubbing and scratching. The children affected are nearly always robust and otherwise healthy, and they are quite as often breast-fed as bottle-fed; and although most of the cases occur at the teething age, many begin earlier.

In rather older children, during the first and the first half of the second decade, small circumscribed patches of scaly eczema are frequently seen about the face. These cases are probably due to improper drying of the face,

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either after washing or more commonly in patients who dribble or have running noses, though streptococcal infection may be responsible for some cases. They are common in children with adenoids. The term *pityriasis simplex* is sometimes applied to this condition.

In adult life the most common forms of eczema of the face and scalp are of seborrhoeic origin, and are considered under *SEBORRHOEIC DERMATITIS*, but, apart from these, the erythematous type is not uncommon. It is more frequent in middle-aged and elderly persons, but may occur at any time of life, and is very frequently secondary to eczematous lesions elsewhere, particularly to an infected eczema of the leg, though cases may occur in which there are no other skin lesions. Usually acute in its onset and course, it is liable to recurrence.

In some cases the patient may wake up one morning and find the whole face red and swollen, the eyelids intensely cedematous and difficult or impossible to open. The scalp is rarely attacked, but often the dorsa of the hands and forearms are affected simultaneously, and occasionally almost the whole body may be involved. Cases of this type usually run a fairly rapid course, the edema and redness subside, a dry scalliness supervenes, and the skin returns to the normal. Occasionally the condition persists, the skin becoming dry, thick, and rigid, and the creases very pronounced. In more intense cases vesicles and bullae may form and a moist dermatitis supervene; secondary infection then occurs, and in such cases the condition generally takes longer to subside. Often the onset is less acute, red patches forming which gradually spread and involve the whole face. There is a great tendency for eczema of these types to recur.

Another form of eczema is a papular or papulo-vesicular eczema which occurs in the centre of the forehead, chiefly in seborrhoeic males or those who perspire freely. In most cases it is accentuated, if not induced, by the pressure of the hatband, and is predisposed to by the too rigorous use of soap and water. Sometimes it is associated with rosacea. Probably it is not a true seborrhoeic dermatitis.

The scalp is rarely the seat of true eczema, except in the infantile form described above. This is probably due to the protective effect of the hair, and is a strong argument in favour of the external-factor theory of eczema. Practically all the acute inflammatory dermatoses of the scalp are either seborrhoeic or are impetiginous—that is to say, of bacterial origin.

The eyelids are not infrequently the site of a chronic erythematous eczema, which is resistant to treatment and recurrent.

The so-called eczema of the ears is generally an impetigo.

Upper limbs.—The hands and forearms are the areas of the body the most commonly affected, doubtless because they are more liable to injury. The most common form is a papulo-vesicular eruption on the back of the hands and wrists which tends to occur in circumscribed patches. These lesions generally weep fairly profusely, the weeping coming chiefly from scattered, raw, pitted areas, from a pinhead to a lentil in size, scattered through the patch. Secondary infection is the rule, and there is a great tendency for the patches to spread by direct extension, and for fresh patches to occur not only in the neighbourhood but in more distant parts of the body, probably by the mechanism already described. In this type the palms usually escape.

The acute erythematous type, involving the back of the hand and the whole forearm up to or extending a little above the elbow, is met with often in association with a similar condition of the face, and behaves in the same manner.

Chronic scaly patches with little or no underlying redness are also commonly seen, especially about the *knuckles*. Such patches occur, too, in the hollow of the *palm*, and are of a very chronic type, often leading to considerable thickening of the horny layer of the skin and deep fissuring in the creases of the palm (*eczema rimosum*).

The acute vesicular type is seen in its most extreme form on the *palms* (and *soles*) and *between the fingers*. It occurs in patients with excessive sweating of the palms, and was at one time thought to be due to blocking of, and consequent cyst-formation in, the sweat ducts. It is characterized by the appearance, beneath the horny layer, usually rather suddenly, of sago-like bodies, which tend to run together and form clear blisters. Owing to the thickness of the horny layer, spontaneous rupture does not occur, but the blisters as a rule become ruptured by friction. Secondary coccal infection is the rule, and, as many of these vesicles become infected before rupture, lymphangitis and lymphadenitis are not infrequently seen, while they are rare in other infected eczemas. This variety is probably identical with the condition known as *dysidrosis* or *cheiropompholyx*.

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The flexures of the *elbows* and *axillae* are subject to a persistent type of eruption which begins as an erythematous eczema and subsequently tends to weep freely. This variety is considered by many authorities to be of seborrhœic origin. In eczema of the axilla there is a tendency for furunculosis to develop.

Trunk.—Eczema on the body, apart from seborrhœic dermatitis, and that produced by definite irritants such as sulphur or scratching, is comparatively rare as a primary lesion, though the trunk is frequently affected in the more generalized forms. The *nipples* are, however, not seldom attacked, the weeping variety being most common. This is most frequently seen in nursing mothers. Strictly, it is a dermatitis of external origin.

Genitals and anus.—The warm moist areas of scrotum, vulva, and anus are not infrequently the seat of a very intractable form of eczema. On the *anus* and *vulva* lichenification is common, and the lesions are really traumatic, and secondary to pruritus. On the *scrotum* the eczema is normally of a scaly erythematous variety with superficial fissuring, but in many cases weeping occurs. Intense itching, especially at night, is the most pronounced symptom in all these cases.

Lower limbs.—The *legs* are very frequently the site of a troublesome eczema which occurs chiefly in middle-aged and old people, or at any rate becomes progressively more common as age advances. It is associated with chronic vascular stasis; when seen in young adults, it is nearly always the accompaniment of varicose veins, and hence has been called "varicose eczema." The lower third of the leg is usually the starting-point. The attack begins in one of two ways: either as an abrasion, which becomes infected, a spreading dermatitis resulting, often assisted materially by the dressings applied; or as a small dry scaly patch which is intensely irritable and is scratched by the patient and infected. This scaly patch suggests a parasitic origin, and by some authorities has been considered seborrhœic. Once the condition has started, scratching, dressings, stockings, etc., tend to spread the infection, and any secondary variety of eczema may result. Most often it is of the weeping type, generally in an extremely septic condition. Ulceration of a very chronic character frequently occurs. The condition starts on one leg, but usually the other is very soon attacked, probably from contact in bed, and in the advanced cases a more or less symmetrical distribution is seen. As

pointed out above, this type is exceedingly likely to be complicated by lesions in other parts of the body.

The *feet* may be the site of an acute vesicular eczema in the same way as the hands, and in fact are often attacked simultaneously.

The nails.—The nail matrix may be involved in eczema. The nails become thickened and rough, and irregularly formed nail substance grows beneath the free edge.

Diagnosis.—Diagnostically, the eczemas, including the dermatitis due to external irritants, fall into two main groups, the dry and the moist, but this division is not always a clear-cut one, as both conditions may be present simultaneously.

The **acute erythematous type**, especially on the face, may have to be distinguished from *erysipelas*. This latter condition, however, spreads more or less slowly from one spot, has a sharply defined margin, and is accompanied by high fever, while the former usually commences simultaneously in several places, or may even involve the whole face at once; the edge is not sharply defined, and if fever is present it is usually quite slight in amount. *Acute giant urticaria* of the face can be distinguished by the absence of redness; *erythema simplex* by its more general distribution, its transitoriness, and the absence of oedema; *erythema multiforme* by its tendency to occur in small circular lesions, rarely larger than a florin, with no proneness to the formation of diffuse patches, by its marked infiltration, and by usually beginning on the extremities.

The **squamous forms** have in the first place to be separated from *seborrhœic dermatitis*. It must be borne in mind that this dermatitis is a form of "eczema" as defined above, and that it is only on account of its bacterial origin and its special characters that it is not considered under that heading. How it differs from squamous eczema may be seen from the article on the subject. The *ringworms* are diagnosed by their sharply defined scaly margins with a tendency (in most forms) to clear in the centre, by the finding of the offending fungus in the scales, and by the rapidity with which they clear up under certain specific antiseptic remedies. A special form, groin ringworm, is differentiated by its sharp edge and slow spread, by its tendency to occur symmetrically on either side of the *foreskin*, and by the presence of the *epidermophyton*.

fungus in the scales. *Pityriasis rosea* can be distinguished by its more or less acute and generalized onset, by its distribution chiefly on the upper part of the trunk and arms, by the oval shape of the lesions, which are pinkish in colour, and by the curious collarette of scales, with the free edge directed inwards, attached about a millimetre from the edge of the patch. *Psoriasis*, when occurring in isolated patches, may be difficult to distinguish from some very chronic eczemas. In psoriasis the sites of predilection are the knees and elbows, and often a history of a typical attack may be obtained. Itching is generally absent or slight, in contradistinction to eczema. Even the smallest papule has a scale on it, and thereafter scales are laid down from below upwards, like the slates on a roof, and can be removed entire, leaving a dry shiny surface. The scales of eczema are much more irregularly laid, and if removed some oozing generally occurs. When the typical discoid lesions of psoriasis are present no difficulty can arise. *Lichen planus*, in its scaly form, can be distinguished by the presence of the small, angular, purplish or lilac-coloured papules which surround the scaly patches, by the absence of moisture of the lesions, and by the presence of patches on the mucous membranes.

The moist eczemas have to be distinguished from *impetigo contagiosa*. It is not always easy to determine whether a moist dermatosis is a primary eczema or a primary impetigo. A moist eczema, when much infected and crusted, is often spoken of as an "impetiginized" eczema, and similarly when the lesions of an impetigo run together and form crusted plaques we speak of an "eczematized" impetigo. The diagnosis of impetigo may be made by the presence of isolated thin-walled blisters or small circular crusted lesions, by the rapidity with which they clear up under simple antiseptic treatment, and by the absence of the papulo-vesicular lesion so characteristic of eczema. The vesicular eczema of the hands and feet must be differentiated from ringworm. Ringworm fungus has been found in a large number of these cases recently, and it is necessary always to be on the look out for the fungus. All localized symmetrical cases should be suspected, but even the acute symmetrical cases should not be overlooked. The diagnosis can only be made by removing portions of epidermis from the edge of the vesicles and examining them for mycelial filaments in liquor potassae under high power. Several specimens

often require to be examined, as the fungus is not always found readily.

Ringworm fungus also produces a very characteristic fissuring "eczema" between the toes, generally between the little toe and its neighbour, and usually bilateral. As a rule it is possible to make the diagnosis at sight, the thick sodden epidermis surrounding a crack in the interdigital angle being characteristic, but the fungus should always be searched for. In both these groups of cases the fungus usually found is the *Epidermophyton inguinale*.

It must not be forgotten that the parasites of scabies and pediculosis may produce "eczematous" lesions, and it is necessary, therefore, to exclude their presence. In both cases the main lesions are scratched papules, and the eczematous lesions, when present, are mixed up with them. In scabies we find acarine burrows on one or more of the following sites, viz. the genitals, wrists, between the fingers, the anterior axillary fold, the elbows and knees, and the feet, and the scratch-papules form around these burrows; except in children, the face and neck usually escape. In pediculosis vestimentorum the scratch-marks are mainly visible on the shoulder, back, and around the waist, while in the pubic variety they are limited to the pubic, chest, and axillary regions. In the former variety the pediculi or their nits may be found on the undervest, in the latter on the affected hairs. Pediculosis capitis is often responsible for eczema of the scalp and neck, which is generally an eczematized impetigo. The diagnosis is settled by finding numerous nits on the hair and by the presence of pediculi on the scalp.

Prognosis.—The prognosis of dermatitis due to known external irritants is usually good, the dermatitis rapidly subsiding under appropriate treatment as soon as the cause ceases to operate. Some individuals, however, develop a generalized hypersensitiveness, and in these cases the eruption may persist. It is wise, therefore, to give a cautious prognosis.

The facial eczemas in infants are usually persistent, and not infrequently take several months to subside even under the most careful treatment; if they are neglected they may be complicated by a most troublesome form of chronic skin sepsis. Eczema of the leg in people past middle age is also a very chronic affection, and, though it is comparatively easy to get it well with rest and appropriate local treatment, the tendency to recurrence is very great. All eczemas tend to recur, and this is

particularly true of the erythematous type on the face. The chronic fissuring eczemas of the palm and the scrotal eczemas are very resistant to treatment.

Treatment. Prophylactic.—When the dermatitis is due to a known irritant, this must, of course, be avoided as far as possible. That is not always possible in occupational dermatoses, but in the severer recurrent forms it may be necessary for the sufferer to change his occupation. Short of this, local protection may be afforded by the wearing of gloves, veils, etc., together with scrupulous care in removing the irritant after work, while strong additional irritants in the shape of soda and alkaline soaps are carefully avoided. In the dermatitis caused by the too free removal of oil from the skin, as in "washerwoman's" dermatitis, the application of cold cream or a dilute glycerin preparation after drying the hands will be found of great benefit. Eczematous subjects must protect themselves from the cold wind, the hot sun and the fire, all potent exciting causes. If a particular plant is the possible cause of the trouble, it should be removed from the house.

Local treatment.—The cause having been removed, the main factors in treatment are rest and protection of the affected areas. The use of soap and water, especially the former, is for the most part contraindicated. It may be necessary to use water for bathing off crusts, etc., and provided a protective substance is applied immediately, little harm will be done. Protective agents have to be varied according to the variety and intensity of the inflammation. In acute inflammations we have generally to content ourselves with lotions, with or without dressings, as grease is badly tolerated. The lotions most frequently employed are calamine and lead. Calamine lotion is a suspension of 5 per cent. each of prepared calamine and zinc oxide in water; it is usually applied at frequent intervals without a dressing, the powder deposited on the skin when the water evaporates forming a protective layer. It has the disadvantage of drying the skin rather too much, but this can be avoided by the addition of 3 per cent. of glycerin. It is found advisable in some cases to reduce the powder content down to as little as 1 per cent. of each ingredient. This type of lotion is most suitable in acute erythematous cases, and those moist cases in which the discharge is slight and there is little secondary infection. Lead lotion is a 2.5-per-cent. solution of the liquor

plumbi subacetatis fortis (B.P.) in distilled water, and is generally applied on lint or linen constantly kept moist with it. Rectified spirit 2-3 per cent. may be added if it is desired to increase the rapidity of evaporation. Lead lotion protects the oozing surface by producing an insoluble albuminate of lead. It is most valuable in moist cases without much secondary infection. If it produces too great a drying effect it may be made up with milk instead of water. When much sepsis is present, and the lesions are very moist, it may be better to use a stronger antiseptic. Perchloride of mercury 1 in 4,000 is very satisfactory, and acriflavine 1 in 1,000 is very useful, though it has the disadvantage of being a bright yellow colour.

Once the acute stage has subsided it is usually advantageous to resort to an oily preparation, as there is a tendency to cracking of the surface. First preparations containing a mixture of oil and water may be used for the cooling effect of the water and the emollient effect of the oil. The preparations most useful at this stage are the linimentum calaminæ (B.P.C.), or one of the numerous zinc or cold creams. If it is undesirable to use calamine or zinc oxide, the ordinary linimentum calcis, with any appropriate medicament added, is useful. At this stage ichthyol is very valuable, used in 5-10 per cent. strength, or, if there is much itching, carbolic acid 1-2 per cent., or creosote in the same strength, may be added. A little later the use of water can be given up and oils or ointments substituted. These can be obtained in any consistence, either as a liquid oil, or in the linimentum calaminæ composition (B.P.C.), or as an ointment which liquefies on the skin, as ung. zinci, or of firmer consistence, as Lassar's paste. The zinc oxide type of preparation, consisting of zinc oxide and starch 50 per cent., and soft paraffin 50 per cent., is the most useful basis for the application of medicaments, as it forms a firm dressing when spread on linen or lint; it is, however, not suitable if there is much discharge, for it tends to dam back secretions and not infrequently to spread the dermatitis. In chronic dry cases it is invaluable.

When an eczema has reached this chronic dry, or only very slightly moist, stage and will not disappear under simple protective dressings, it is necessary to use mildly irritating applications for "stimulation." Salicylic acid is most frequently employed, as it is antiseptic, keratolytic, and slightly stimulant in its action; it is used in 1-3 per cent. strength. Other

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agents are coal tar 1-5 per cent., oil of cade 5-10 per cent., pyrogallol acid 1-2 per cent. (or, better, the German preparation Lenigallol 5-10 per cent., when obtainable), or chrysarobin 1-2 per cent. In cases which resist these more active agents, radiotherapy is the best treatment; a dosage of 3 Sabouraud pastille at a single sitting is recommended. In some cases, especially the pruritic ones, applications of the mercury-vapour (Kromayer) lamp give excellent results.

Chronic weeping cases, if not very septic, are best treated by painting the surface daily with 1-2-per-cent. silver nitrate dissolved in sp. ætheris nitrosi, afterwards covering it with lint soaked in lead lotion or with zinc paste.

In chronic septic cases it is necessary to remove the crusts. This may be done by soaking in warm water or warm oil, or, in cases where they are very thick or adherent, by the application of starch-and-boric-acid poultices. After the crusts have been removed, zinc ointment or zinc paste containing a weak mercurial such as hydrargyrum ammoniatum or hydrargyri oxidum flavum 2-3 per cent. should be applied.

When there is much thickening of the horny layer, salicylic acid is the most useful application; it can be used either as a paste or as a plaster. The emplastrum salicylicum compositum (B.P.C.) is a useful preparation which contains 20 per cent. of salicylic acid.

Crude coal tar is a very valuable remedy in chronic non-septic cases. It is painted on over the affected area and allowed to dry, the surface then being dusted with talc powder to prevent the clothes from sticking to it. It forms a complete protective layer and prevents movements of the skin, and is also an antiseptic and antipruritic. It should never be used in septic cases, as it prevents the discharge from escaping, but a certain amount of serous discharge does not appear to contraindicate its use. If it is to be applied over a very mobile part, such as the flexure of the elbow, it is advisable to use a splint.

General treatment.—In the early stages of dermatitis due to a known irritant, little or no general treatment is required. If, however, in such a case, generalization occurs, the health suffers, and rest, regulation of the bowels, and appropriate diet are necessary, together with general tonics. In cases of which the origin is doubtful, a careful examination is necessary to detect digestive disturbances or other functional irregularities. Diet should be light. In the more acute cases it may be advisable to put

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the patient on a rigid milk diet; in other cases sugars and starchy foods should be reduced to a minimum, and hot and highly-seasoned dishes, soups, shell fish, salted meats, and cheese forbidden. Alcohol, strong tea, and coffee should also be stopped, and meals should be taken without much fluid; half a pint of hot water night and morning, and half an hour before meals, may be recommended. An alkali such as sodium bicarbonate may be given after meals. Constipation must be dealt with either by saline aperients or, in the more troublesome cases, by cascara, senna, aloes, or podophyllin. Intestinal fermentation may be met by the administration, three times a day, of salol or salicylate of bismuth 10-15 gr., ichthyol 3-5 min., or menthol 1-2 gr., in gelatin capsules, or liq. hydrargyri perchloridi 1 min.

If gout is obviously present, alkaline waters or colchicum should be given.

In debilitated subjects cod-liver oil with or without malt is often of great value, while in anæmia iron or arsenic may be administered. In the more florid type of case vin. antimon. in 5-min. doses three times a day is useful.

For the wealthy, spa treatment is often beneficial in the more chronic and recurrent cases, the waters of Aix-les-Bains, Harrogate, Strathpeffer, Contrexéville, Vichy, and Cheltenham being most suitable for the gouty and florid types, while those of Spa, La Bourboule and Royat are good for debilitated and anæmic patients.

In acute cases, rest in bed is of great importance, not only for its sedative effect on the nervous system, but also because it protects the skin from the changes of temperature and exposure to various irritants. Sleep is a prime necessity in all cases, and this may necessitate the giving of sedative drugs to alleviate the irritation, and also of hypnotics. Of the former, bromides are the most useful, while in the latter, sulphonal, trional, and chloral hydrate may be prescribed. A. M. H. GRAY.

ECZEMA MARGINATUM (see RINGWORM).

ELECTRIC DISCHARGES (see LIGHTNING AND ELECTRIC DISCHARGES, INJURIES AND DEATH FROM).

ELECTRICAL REACTIONS.—The chief forms of electricity that have been used in the investigation of the electrical reactions of muscles and nerves are the galvanic and faradic currents.

Normal reactions.—If a galvanic current

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of a few milliamperes' strength be applied to a healthy muscle by means of a small disc electrode placed in contact with the skin over it, the muscle contracts with a sharp twitch at the instant the circuit is closed, and then during the continued flow of the current through it appears quiescent. If the current be of sufficient strength, however, there may be another twitch at the instant its passage is discontinued. These opening and closing twitches may be obtained if either the kathode or the anode be used as the electrode over the muscle, for, although physiological experiments show that the kathode is the exciting electrode at the make of the circuit only, and the anode at the break only, it is impossible in the conditions that obtain in clinical muscle-testing to get absolutely unipolar effects underneath the so-called active electrode.

There are, therefore, four possible contractions—the kathodal closure and the kathodal opening contractions, and the anodal closure and opening contractions. These are usually designated by the abbreviations KCC, KOC, ACC, and AOC. They are not all obtained with the same minimum strength of current, nor are all of the same magnitude from a current sufficient to produce them all. The one most readily obtained is the KCC, and the order of appearance is KCC, ACC, AOC, and KOC. It is much more difficult to obtain the opening contractions than the closing ones, and, for the former, painful currents are frequently required. It is customary, therefore, in clinical practice to pay attention to the closing ones only.

The active electrode may be placed on the skin over the muscle, or over the trunk of the nerve supplying it if the latter is near the surface. In the one case the responses constitute the muscle reactions, and in the other the nerve reactions. When the electrode is applied over the muscle the best results are obtained by placing it over the motor point, or point of entry of the nerve into the muscle, and it is probable that the stimulus usually reaches the muscle-fibres mostly by way of the intramuscular nerves, which respond more readily to the current than does the less excitable muscle-tissue. The latter, however, is capable of direct excitation apart from nervous intervention, provided the duration of the current be long enough.

If the **faradic current** be used as an excitant instead of the galvanic, a sustained contraction

or tetany is produced in the muscle during the time it is passing, instead of a **make-and-break twitch**. The ordinary faradic current has a frequency of thirty to forty interruptions a second, and this tetany represents the summation of a series of shocks sent into the muscle in such quick succession that no interval of relaxation occurs between the individual contractions. If the interruptions be slowed down to one or two a second, separate contractions may be obtained. The faradic current probably differs from the galvanic, therefore, chiefly in the short duration of the stimulus of its individual components. The galvanic excitation consists of a long wave stimulus, the faradic of a series of short waves.

Abnormal reactions.—A diminution in the excitability of the nerve and muscle may be observed in cases of fatigue, in the slighter degrees of peripheral nerve lesions, in the slow wasting of muscles due to chronic degenerative diseases of the lower motor neurones, in some old cerebral palsies, and in the wasting of muscles associated with disuse and with joint-lesions. An increase of excitability may be found in the early stages of more severe acute lesions of the lower motor neurone, in some recent cerebral palsies, and, as a general rule, in many conditions accompanied by increase of the tendon reflexes. Simple quantitative alterations in the excitability occur, therefore, in many varied conditions. In their slighter degrees they are difficult to detect and may not be of much significance.

More important alterations are observed in what is called the **reaction of degeneration**. In this condition the excitability of the nerve to both faradism and galvanism is lost entirely; the excitability of the muscle to faradism is also lost, but is preserved or even increased to galvanism. The contraction produced by the galvanic current, however, is a slow, sluggish one, instead of the usual short, brisk twitch. Sometimes, also, it shows a polar change, so that the KCC, instead of being greater than the ACC, is equal to, or less than, the ACC. Usually, too, a better contraction is obtained by placing the active electrode over the tendon end of the muscle rather than over the motor point. This last phenomenon is called **longitudinal hyperexcitability**, or the **longitudinal reaction**. It is due to the fact that, as the sluggish contraction results from the direct stimulation of the muscle-fibre, this stimulation, in the absence of nerve excitability, is likely to be more effective when the

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current traverses the whole length of the muscle.

The development of these changes is often gradual, and it may be a week or ten days after the onset of the lesion before they are fully established. During this time the gradual loss of faradic and galvanic excitability in the nerve, and of faradic excitability in the muscle, may be preceded by an initial slight hyperexcitability for a day or two. The reaction of degeneration may be regarded, therefore, as a syndrome corresponding to certain stages in the affection of the muscle and nerve. If the lesion be more severe or progress still further, the galvanic excitability of the muscle diminishes and all response to both currents in both muscle and nerve is lost.

According to the severity of the lesion there may be stages between simple quantitative diminutions of excitability and the complete reaction of degeneration, and hence arise cases of **partial reaction of degeneration**. This term, as usually understood, refers to conditions of response in which there is more or less preservation of faradic and galvanic excitability in the muscle and nerve, and even a hyperexcitability in the muscle; while the contractions obtained by stimulating it with galvanic current show the qualitative changes, the sluggishness, the polar changes, and the longitudinal hyperexcitability characteristic of the typical reaction of degeneration.

The reactions of degeneration, complete and partial, are obtained in those affections of the lower motor neurone that are acute or sudden in onset. They are well seen, therefore, in such diseases as acute poliomyelitis and the traumatic lesions of the peripheral nerves, but not in the slow degenerative diseases of the lower motor neurone such as progressive muscular atrophy and other chronic spinal conditions, or degenerative nerve-lesions, or primary muscular dystrophies. In these affections electrical testing is more likely to reveal simple quantitative changes, a gradual diminution of faradic and galvanic response, ending finally, as the wasting proceeds, in absence of all response.

Certain special reactions have been described in different diseases, such as the myasthenic reaction, the myotonic reaction, Rich's reaction. In the **myasthenic reaction**, which occurs in myasthenia gravis, the faradic excitability can be quickly exhausted by repeated stimulation with a series of separate shocks, the galvanic excitability remaining unaltered. After a few minutes' rest the faradic excitability returns.

The repeated stimulation of the muscle causes the same temporary exhaustion of it as does repeated voluntary use of it in this disease. In the **myotonic reaction**, met with in conditions of myotonia, as for example in myotonia atrophica, the contraction on faradic stimulation persists after removal of the stimulus; that is, the muscles, once contracted, relax slowly, as they do in myotonia atrophica after voluntary contraction. In **Rich's reaction** the KOC is obtained with abnormal readiness. It was thought to occur in cases of compression of nerves.

The ordinary uses of electrical muscle-testing are in the differential diagnosis of functional and organic paralysis, and in the estimation of the extent and severity of the lesion in many cases of lower-motor-neurone disease or injury. In those lower-motor-neurone affections of acute or subacute onset in which reactions of degeneration are liable to occur, such as poliomyelitis, traumatic lesions of the spinal motor nerve-roots, brachial-plexus lesions, and peripheral nerve injuries, a knowledge of the presence or absence of abnormal electrical responses is particularly valuable and affords information for the early diagnosis of the extent of the affection and for an estimation of its prognosis. Mild cases of a facial palsy, for instance, may show no electrical change or only a slight quantitative alteration, intermediate cases a partial reaction of degeneration, severe cases a complete reaction of degeneration, while still worse cases may give no electrical response at all.

In the electrical treatment of cases of paralysis of this kind, a knowledge of the electrical responses in the affected muscles is of help in deciding what current should be used for their stimulation, and in estimating from time to time their progress. It is of great value, too, especially in peripheral nerve lesions, in deciding as to the advisability of nerve suture or other operative procedure that may be necessary if the conductivity and excitability of the nerve are completely destroyed.

Technique.—In the testing of reactions it is convenient to have the two currents available in the form of a combined switchboard supplied with a galvanometer in the galvanic circuit and a current reverser. In the unipolar or usual method of testing, the active electrode is a small disc which may have a make-and-break key in its handle, and the indifferent electrode is a large flat one. The active electrode is placed over the motor point of the

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muscle or nerve, the indifferent one on some distant part of the body. In the bipolar method, which may be of value where the excitability is much diminished, the two electrodes are small ones and are placed over the muscles and nerves to be examined. The electrodes and the skin underneath them should be well wet with saline solution or tap-water, and the electrodes firmly and evenly pressed on the skin during testing.

The faradic current is employed first, then the constant current. In using the latter it is necessary to note the reading of the galvanometer with which the contraction is obtained and the character of the contraction, to compare it, if possible, with that obtained from the corresponding muscle of the sound side, to contrast the effects of the kathode and anode as the active electrode, and to test for the longitudinal reaction. The examination must be carried out with the active electrode not only over the individual muscles, but also over the main nerve-trunk if that be accessible, and, in cases of nerve injury, above and below the site of injury if that be possible.

The classical testing of electrical responses by the faradic and galvanic currents is open to objection on the ground that the use of these currents is lacking in scientific precision. The faradic current as produced by the ordinary induction coil is an uneven, irregular current with many varying factors in its composition. The results of testing with it are only roughly comparable in different cases, and no accurate differentiation of lesions can be made. To the galvanic current, as ordinarily used, it has been objected that the chief feature of the reaction of degeneration—namely, the slow, sluggish character of the contraction—is one the estimation of which depends on the personal observation and experience of the examiner, hence the standardization of results is impossible.

Various attempts have been made to introduce more scientific and accurate methods, as, for example, by the use of galvanic currents of measured strength interrupted by mechanical devices instead of the induction coil current, and by the use of condenser discharges through the muscles instead of faradic and galvanic currents, in order to produce contractions. As the duration of a condenser discharge is proportional to its capacity, it is possible by using condensers of different capacities to obtain waves of short and long duration corresponding to the faradic and galvanic waves.

The different capacities may be obtained

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either by charging a set of suitable condensers at a variable potential, or by using a series of different condensers charged at a constant potential. The latter is the method that has been most used in clinical work. A testing-box containing a series of condensers of appropriate capacities charged at a constant voltage is arranged so that an ascending series of capacities can be discharged by means of electrodes through the muscles to be tested. The presence or absence of a contraction is alone what is looked for, and the minimal capacity required to produce it in the different muscles investigated is noted. They can thus be compared with one another and with the normal.

Notwithstanding the advantages claimed for condenser-testing, the old method still holds its own. Its use has been established by long custom, and the correlation of its results with clinical data and their interpretation in clinical terms are widely known. P. W. SAUNDERS.

ELECTRICAL TREATMENT.—The applications of electricity in medical treatment are very numerous, and it will be impossible to describe them here in detail. Some of the more important uses of the different forms of current electricity, and of static or frictional electricity, may be considered briefly.

The forms of current electricity of therapeutic importance are the galvanic, faradic, sinusoidal, and high-frequency currents. The **galvanic or continuous current** is used for its effects on the tissues generally, or for its effects locally on some particular part of the body. For its more general action it is used in the full-length bath, in the four-cell Schnée bath, or by means of ordinary electrodes in the methods of general or central galvanization, in which one electrode is kept stationary on the buttocks or abdomen and the other moved about over different parts of the body, so as to include large areas in the treatment. General applications of this kind are employed for their tonic and sedative effects in various conditions—in anæmia, in debility or convalescence after acute illness, in constitutional diseases, in chronic sciatica, lumbago, or “muscular rheumatism,” in multiple neuritis, sometimes in chronic cerebral palsies, and in neurasthenia and the insomnia associated with it.

Localized applications are usually carried out by means of ordinary electrodes in *stable*, *labile*, or interrupted methods, or by the use of small limb-baths with or without interruptions in the circuit. *Stabile applications*, in

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which the electrodes are kept stationary and the current passes evenly through the tissues, have a sedative nutritional effect, and are used chiefly in painful sensory-nerve conditions such as sciatica or brachial-plexus neuritis, or in irritable motor conditions such as ties and spasmodic movements of muscles. Labile and interrupted applications, in which the current passes through the tissues intermittently, have a more stimulating effect. They are used mostly in cases of flaccid palsy such as occurs in poliomyelitis and peripheral-nerve lesions, and in the form of baths in various vaso-motor affections such as Raynaud's disease or chilblains, or the cold, blue condition of the extremities that frequently accompanies lower-motor-neurone paralysis.

In all these methods of treatment the kathode as the active electrode is usually the more stimulating and the anode the more sedative. Anodal stable galvanism is sometimes called sedative galvanism.

The passage of a continuous current through the body is accompanied by a movement of ions, comparable to that which takes place in its passage through an aqueous solution of a salt, and it is possible that the chemical changes associated with this ionic movement may be responsible for the nutritional results that are ascribed to galvanic treatment and which show themselves in mildly stimulant or sedative effects, or what are sometimes called the "refreshing" effects of the current on the tissues. The chief results, however, produced in these general and local applications probably come from the action of the current on nerves and muscles and vaso-motor tissues.

The direct effect of the current on nerves and muscles is probably greater than on other tissues of the body. We know from physiological experiment that the passage of the current along motor nerves is accompanied by an alteration of their excitability and conductivity in the region of the poles, that this alteration is different at the two poles—katelectrotonus or increased excitability obtaining at the one, and anelectrotonus or diminished excitability at the other—and that, in addition to these accompaniments of the even passage of the current, there may be certain shock effects at the opening and closing of the circuit which show themselves in contractions in the muscles. The occurrence of these shock effects suggests the more stimulating effects of labile and interrupted applications of the current as compared with the stable, whilst the polar

differences suggest the different values already mentioned in the two electrodes.

It is in the treatment of paralysed muscles that these stimulating shock effects and polar changes become of the greatest value. When the current is applied interruptedly to a paralysed muscle or its nerve, it leads, unless the muscle or nerve is too badly damaged to respond, to a series of contractions which tend, owing to the passive exercise they cause, to promote the metabolism and maintain the tone and nutrition of the muscle. Its use is more hopeful in the affections of acute onset, such as poliomyelitis, where the nervous lesion is likely to subside, than in the chronic degenerative wastings, such as progressive muscular atrophy, where it tends to increase. The interruptions in the current, however, must not be so frequent as to lead to fatigue of the muscles or interfere with the proper renewal of their blood supply.

The electrolytic action accompanying the passage of the current through the body results in the accumulation of alkaline substances such as caustic soda at the kathode, and acid substances like hydrochloric acid at the anode. These substances are partly responsible for the burning and redness of the skin that occurs underneath the electrodes if proper precautions are not taken, and they account for the caustic action of the poles, and especially of the kathode, in electrolytic treatment for the removal of hairs and naevi. In this treatment fine needle electrodes are passed into the hair-follicle or into the tissue to be destroyed.

The electrolytic effects of the current are used also in ionization treatment to introduce drugs into the tissues. (*See IONIZATION.*)

The essential features of the **faradic or induction-coil current** in treatment are its stimulating shock effects on the tissues, and especially on motor and sensory nerves. Unlike the galvanic current, it causes little or no ionic movement, or electrolytic or polar effect. Owing to this absence of electrolytic action, the faradic current can be applied to mucous membranes much more readily than the galvanic, as in the method of treating constipation by means of one electrode in the rectum and the other on the surface of the abdomen.

For its general stimulant effect the current is used mainly in the form of general faradization, a method similar to that of general galvanization. Applied in this way from an ordinary coil, it causes muscular contractions and stimulates the sensory nerve-endings in

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the skin over wide areas, and is useful in cases of neurasthenia and in functional nervous conditions. Another form of general application is by means of the *Bergonié apparatus*. In this a special coil is employed that produces maximum muscular effects with little sensory stimulation, and the treatment is used for the general metabolic effects resulting from diffuse muscular contraction. It is especially advocated in cases of obesity.

Localized applications of the faradic current are used in various disorders. In limb-baths it provides a more stimulating form of treatment than the galvanic current for such vaso-motor affections as Raynaud's disease or chilblains; in ordinary labile applications it acts as a counter-irritant in conditions such as neuralgia and paræsthesia and functional sensory disturbance; while in hysterical conditions of all kinds its effect on the skin makes it a useful aid in treatment by suggestion. In these conditions the greatest skin effect is obtained from the uneven, irregular contact of a wire brush electrode.

In the lower-motor-neurone affections, in which reactions of degeneration are liable to occur, faradic treatment may be used for the paralysed muscles if they still react to faradism. The short duration of the stimulus from the individual currents that make up the faradic series may not be long enough, however, to provoke a contraction in many cases, and, if the affected muscles have lost their faradic reaction, labile galvanic treatment is to be preferred, or a combination of the two currents joined together in series, **farado-galvanism** or **combined treatment**. It is claimed for this latter treatment that the faradic stimulation has a beneficial effect though no visible contractions in the muscles may result from its employment.

When faradism is used for its stimulating effect on paralysed muscles, it is applied in interrupted fashion in the ordinary labile methods, or by the use of some mechanical interrupter in the circuit, or by moving the iron core in and out of the primary coil as in the method of treatment known as "surging" faradism. The object of the intermittent application is to allow intervals of relaxation and rest, as the faradic tetany causes reduction of the circulation and fatigue of the muscle.

The **sinusoidal current** differs from the faradic in the even, regular periodicity of its components and in their long wave-like char-

acter. Its effects are very similar to those of the faradic current, and it has a great effect on the skin. Its chief use is, in the form of baths, to supply a more stimulating treatment than the galvanic current. It may be employed, therefore, in the full-length bath or in small limb-baths in most of the conditions for which galvanic-bath treatment is advocated. In the local limb-baths an interrupter may be used in the circuit to the patient; these local treatments are of especial value in vaso-motor affections and in cases of paralysis in the limbs accompanied by vaso-motor changes. The sinusoidal current is not applied with ordinary electrodes for muscle stimulation, because of its painful effects on the skin.

The **high-frequency current**, like the faradic and sinusoidal currents, is an interrupted alternating current, but it has peculiar properties dependent on the extraordinary speed with which its interruptions occur. It is used chiefly by the *effluve method*, a multiple-point electrode connected with one end of the Oudin's resonator of the high-frequency machine being passed quickly near the surface of different parts of the body, so as to produce a breeze or sparks between it and the body. The other methods of treatment, by condenser couch, auto-conduction, or ordinary electrodes, are not much employed.

Applied by the effluve method from an ordinary Leyden jar machine, the high-frequency current stimulates the sensory nerve-endings in the skin and produces a dilatation of the superficial blood-vessels, and it is said therefore to reduce blood-pressure and increase metabolic processes generally. It has also a considerable psychic effect on hysterical patients.

The high-frequency current as produced by the Leyden-jar oscillatory discharge, however, is one in which the component currents are gradually damped down, and cease altogether at recurring intervals. If they can be maintained steadily at the high-frequency rate they produce a deep heating effect along their passage through the body. (See DIATHERMY.)

Superficial heating from ordinary electric currents is obtained in **radiant-heat baths** of different kinds, local and general, in which the heat of carbon lamps is used to raise the temperature of the surface of the body. Applied in large or full-length baths, this provokes sweating and influences metabolism generally; applied locally, it relieves pain in joints and muscles in various rheumatic and other con-

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ditions. Other heating effects from electricity are used in the electric cautery.

Static electricity is obtained for treatment purposes from a Wimshurst machine. It is employed in various ways. In the method of simple charging, the patient sits on the insulated stool and holds the ring electrode connected with one or other pole of the machine, usually the positive. While charged he may be discharged by having a multiple-point crown electrode, earth-connected from the other pole, placed near his head so that he feels a breeze from it, or he may be more abruptly discharged by means of a knob or point electrode brought quickly past some part of his body, so that he feels a sudden spark or the sensation of a severe blow.

Simple charging has a general sedative effect, as also has the breeze applied to the head. These methods of application are used in the treatment of neurasthenia, and the breeze is especially valuable in treating the insomnia and headache of that condition. Static applications are said to raise the blood-pressure, however, and are contraindicated, therefore, in neurasthenia associated with high blood-pressure.

The spark treatment is the most vigorous method of static application, and is of value as a counter-irritant treatment for local persistent pains of psychical origin, or even for the pain of a chronic neuritis, such as a long-standing sciatic pain due to adhesions about the nerve. Possibly the sudden ionic displacement and sudden local muscle movement caused by the spark may break down the adhesions.

Static electricity is not used for local applications to muscles, except in condenser form. Small condensers charged to a known potential have been used in testing and treating paralysed muscles. The condenser discharge through the muscle provokes a contraction and serves as a stimulant to preserve the metabolism of the muscles and prevent their degeneration in lower-motor-neurone lesions.

A brief reference may be made to the use of electricity in the treatment of hysterical manifestations. In these it may make little difference what form of electricity is used, but the psychical effect will probably be greater the more elaborate the apparatus and the more intense the skin effect it produces. A high-frequency treatment with sparks, or a faradic wire-brush treatment, will have more effect, as a general rule, than an even stable galvanic application.

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In the treatment of local hysterical palsies the faradic current is the one most used, because of its skin effect and because of the ready control which it enables the physician to exercise over the paralysed muscles, and the ability it gives him to cause contractions in them at will, or to aid the patient's efforts to produce a voluntary contraction. It is of use, therefore, not only as an adjunct to suggestion, but as a means of demonstrating to the patient the possibilities in his muscles, and as an aid in re-educating him in their use.

P. W. SAUNDERS.

ELECTROCARDIOGRAPHY (*see* HEART-BEAT, ABNORMALITIES OF).

ELEPHANTIASIS (*see* FILARIASIS).

EMBOLISM, CEREBRAL (*see* CEREBRAL VASCULAR DISEASE).

EMBOLISM OF MESENTERIC VESSELS (*see* MESENTERIC VESSELS, EMBOLISM AND THROMBOSIS OF).

EMBOLISM, PULMONARY (*see* LUNG, EMBOLISM OF).

EMPHYSEMA.—Destruction of the walls of the pulmonary alveoli, causing loss of their elasticity and of the aerating and vascular surfaces which they contain. It may occur with enlargement of the lung, or may be atrophic and accompany the general atrophy of old age. The former variety is that usually understood by the term *emphysema*; the latter is not commonly met with in a very marked degree. "Compensatory" *emphysema*, which complicates other pulmonary diseases, is considered in connexion with those diseases.

HYPERTROPHIC EMPHYSEMA

Etiology.—Hypertrophic *emphysema*, in which the lung is large, affects those who need to make prolonged expiratory efforts from any cause, and especially when their work is in the open air. It is therefore common among navvies, watermen and agricultural labourers. It is found in those who frequently suffer from a cough or who earn their living by the use of wind instruments. A temporary condition of over-distension may follow an acute illness in which cough is a factor, such as whooping-cough.

Pathology.—The upper lobes and the parts of the lower lobes that are not supported by the chest-wall are chiefly affected; *emphysema* may therefore be found in the margins of the lower

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lobes. In the upper lobes the apex and free margins show the greatest changes. It sometimes appears in lines corresponding to the intercostal spaces. In advanced cases the lungs may be emphysematous throughout. The parts affected are white, the markings of the surface are more distinct than usual, bullæ are formed, and air can be squeezed from one part of the emphysematous area to another. The lung is doughy in consistence, does not collapse, and floats high in water. Microscopically, it is seen that the elastic tissue has disappeared and that the walls of the air-vesicles are largely obliterated. There is thus loss of elasticity, loss of the vascular sheet, and loss of the aerating surfaces. In advanced cases the right heart is dilated and all the concomitant effects of right-heart failure may supervene.

Symptomatology.—Shortness of breath on exertion, accentuated when the air is dry and cold, is the chief symptom. Chronic cough is also common. The shape of the chest resembles that of the upper half of a barrel, and the epigastric angle is very broad. During inspiration there is little expansion of the chest. Epigastric pulsation is obvious. The face and hands are congested and cyanosed and the conjunctivæ injected. The abdomen is generally prominent. Expiration is prolonged and performed with great effort. There is discomfort on lying down. The expiratory murmur may be longer than normal or may be inaudible. Dullness indicating dilatation of the right auricle may be detected to the right of the sternum. The patient frequently suffers from chronic bronchitis, which chiefly affects the lower lobes, and râles and rhonchi are often heard.

Diagnosis.—This should offer little difficulty. The shape of the chest, the laboured expiration, and the general appearance are quite characteristic.

Prognosis.—In favourable circumstances the patient with emphysema may live for a long time, provided that he can afford a quiet life, does not suffer from attacks of bronchitis, and there is no severe concomitant condition. Heart failure and chronic bronchitis are dangerous complications.

Treatment.—A person with hypertrophic emphysema is usually too stout, and the secret of treatment is to reduce this corpulence and prevent its recurrence. Drastic measures have to be adopted with this object. The chief items of the diet to be omitted are those

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containing starch and fat. After the introduction of a diet consisting of meat and fish, green vegetables, fruit, and a small quantity of toast or biscuits, a day's fasting should be ordered every other day. During this period it is advisable that the patient should take his usual exercise, which may be slightly diminished on the fast days. When a considerable amount of weight has been lost, the shortness of breath will be greatly ameliorated. Having attained a satisfactory condition, the weight being not greater than it was at the prime of life, the patient must be ordered to maintain the reduction. Any increase of weight must be met by a proportionate decrease of diet.

Various methods of treatment, as by chest compression or breathing into a rarefied atmosphere or against increased pressures, have been tried, and may produce some temporary improvement.

The same care should be exercised to prevent patients catching cold, etc., as in cases of bronchitis and bronchiectasis (q.v.), and should a liability to bronchitis be present, the prophylactic measures suitable for that disease should be observed. Any indication of cardiac failure must be treated by rest in bed.

When bronchitis develops, it requires its appropriate treatment. Bleeding to 10 or 20 ounces is of great value. Potassium iodide is especially indicated for the emphysematous in whom bronchitis is persistent.

ATROPHIC EMPHYSEMA

The atrophic form is generally associated with a small chest, which is fixed and does not expand. Secondary cardiac changes are absent. As this condition usually occurs in the aged, the symptoms are those of senility, and its presence is rarely detected.

CHARLTON BRISCOE.

EMPHYSEMA, SURGICAL.—The presence of air or gas in appreciable quantities in the subcutaneous and cellular tissues of the body.

Etiology.—There is usually a breach in the walls of an air-containing cavity or viscus, such as the lungs, the trachea, the nasal cavity and adjacent sinuses, or the mastoid cells, and some force to propel the air into the tissues.

By far the most common cause is injury of the lungs due to a fractured rib. Both layers of the pleura are generally injured, yet in most cases no pneumothorax is demonstrable.

The amount of air which escapes is very variable, and the full extent may not be noticeable until a day or two after the injury. It may extend up to the neck or face, or down to the loins or pelvic region, and in exceptional cases may even encroach on to the limbs.

Occasionally, vesicles of the lung rupture and allow the escape of air under the visceral pleura; this air may travel up and appear at the root of the neck. Exceptionally, air may enter the tissues from without when there is a valvular wound of the chest-wall.

Surgical emphysema beginning in the neck may result from a tracheotomy or other wound of the trachea. Fracture of the nasal bones or implicating the walls of the frontal sinus may be accompanied by a breach of the lining mucous membrane, so that when the patient blows his nose air escapes into the subcutaneous tissues of the forehead. The same act may cause air to enter the orbit and produce a sudden exophthalmos in some cases of nasal sinusitis if the bony partition between the nose and the orbit is eroded.

The term "surgical emphysema" is also applied to the condition caused by the escape of gas from the intestine when it is ruptured behind the peritoneum. In the absence of fractured ribs, emphysema noticed first in the abdominal wall would suggest intestinal rupture.

Diagnosis.—The affected tissues are somewhat swollen, and when the flat of the hand is pressed gently over the area which contains air a characteristic crepitant sensation is experienced; when once this has been felt it can never again be mistaken. The only other condition causing gas in the tissues is *emphysematous gangrene*, which is accompanied by other unmistakable signs and symptoms.

The **prognosis** of surgical emphysema itself is not serious. Unless the primary lesion is fatal the air is gradually absorbed, and within a few days no trace of it can be discovered.

No special **treatment** need be directed to the surgical emphysema, but the condition which led to it needs to be corrected. Fractured ribs need strapping to limit the thoracic movements. Patients who have a fracture of the nasal bones must be cautioned against trying to expel blood from the nose by blowing it violently. Surgical interference is unnecessary unless there be some complication, such as a hæmopneumothorax, and even then only in special circumstances. ZACHARY COPE.

EMPHYSEMA.—A collection of pus in the pleural cavity.

Etiology and pathology.—An empyema, like a simple acute pleurisy, may be set up by almost any of the pathogenic germs, but while some of them, such as the tubercle bacillus and the *Diplococcus rheumaticus*, almost invariably give rise to a simple pleurisy, others, such as the pneumococcus and the streptococcus, produce, as a rule, a purulent inflammation. The vast majority of empyemas are due either to pneumococci or to streptococci and staphylococci. These give rise to what are really two distinct diseases, differing commonly both in symptomatology and in prognosis.

The pneumococcus is the cause of most empyemas in children, and also of a large minority, possibly a majority, of those occurring in adult life. The disease usually follows an attack of acute pneumonia, and the younger the patient the more likely is it to supervene. A pleural effusion after pneumonia is not necessarily purulent, but it is usually, and in young children almost invariably so.

Empyemas due to streptococci, more rarely to staphylococci, or to both combined, occur for the most part in adults, often as part of a general septicæmic or pyæmic process, or along with a severe form of one of the acute specific fevers, especially influenza and scarlet fever. They may also be set up by extension of a virulent inflammation from adjacent parts—from the lung in septic infarction, gangrene, etc.; from the abdomen as a result of a subphrenic abscess, puerperal peritonitis, appendicitis, or tropical hepatitis. The empyemas not uncommonly associated with osteomyelitis in children are due to a pure staphylococcal infection. A penetrating wound of the chest may also produce an empyema.

The tubercle bacillus alone very rarely gives rise to a purulent pleurisy. It is true that pulmonary tuberculosis is by far the most frequent cause of pneumothorax, and that this is often followed by pyo-pneumothorax, but the pus is probably always due to an associated infection, usually streptococcal.

Symptomatology.—This varies enormously in different cases, and although almost any of the causal microbes may give rise to symptoms of any degree of severity, yet for the most part pneumococcal empyemas run a much milder course than the streptococcal and staphylococcal varieties. Dealing first with the *pneumococcal* forms, we note that almost invariably

the patient has just had an attack of acute pneumonia, but there is no necessary relationship between its severity and the subsequent tendency to empyema. Sometimes pus forms before the crisis of the pneumonia has occurred, and then the fever of the latter passes without perceptible break into that of the empyema, merely becoming more intermittent in type. In other cases there is a definite crisis, followed almost immediately by irregular fever; whilst in yet other instances the temperature remains absolutely normal for several days, so that even for a week or longer the patient seems to be making an uninterrupted recovery, and then pyrexia suddenly begins. With the fever the patient shows evident signs of general malaise; there are wasting, loss of appetite, increasing pallor, occasionally sweating and diarrhoea, and the skin may assume a yellowish tint. The pus may form with surprising rapidity, two or three pints, perhaps, accumulating in a few days. It is important to recognize, however, that when the pus is small in quantity and due to a comparatively non-virulent type of pneumococcus, there may be little or no rise of temperature and the constitutional symptoms may, at any rate for some time, be exceedingly slight. In fact, the only indications of the presence of pus may be that the physical signs of consolidation in the lung, instead of clearing up, are to an increasing degree replaced by those of a pleural effusion, and that the child is obviously not improving.

In the *streptococcal* and *staphylococcal* forms the constitutional symptoms are usually much more severe, although it must be remembered that they may be due, in part at least, to a more widespread general infection. The temperature is often of an extremely hectic type, sometimes with rigors. Occasionally it is subnormal—an indication, perhaps, that the patient's resistive powers are quite overwhelmed. Sweating and diarrhoea may be severe; the aspect rapidly becomes one of marked cachexia; and the patient may pass into a well-marked "typhoid state." In some of these cases the pus is foetid, especially when the primary disease is gangrene of the lung or a subphrenic abscess.

In all cases of empyema, whether pneumococcal or streptococcal, the local respiratory symptoms are generally slight; there may be little or no cough, pain, or expectoration. Some dyspnoea is commonly present, but so long as the patient is lying quietly in bed it may not be at all obtrusive.

The physical signs of empyema are practic-

ally identical with those of a simple effusion; we note especially loss of vocal fremitus, displacement of the heart, pronounced dullness and resistance to percussion, weak or absent breath sounds and diminished vocal resonance. Are there any signs which serve to distinguish between the two conditions? There are a few, but unfortunately they are far more frequently absent than present. A pulsating empyema is met with very occasionally; it is always a large one, and on the left side. Edema of the chest-wall and bulging of the intercostal spaces are almost invariably due to a purulent effusion, but are altogether exceptional. Clubbing of the fingers may develop if an empyema is left undrained for a few weeks. Eventually, as a rule, an untreated empyema either bulges externally or ruptures internally. In the former case the pus may point anywhere, most commonly in the fifth intercostal space in the nipple line. Internally it may rupture into the lung, peritoneal cavity, or stomach; sometimes it burrows behind the diaphragm and simulates a lumbar or a psoas abscess. The most frequent site of rupture is into the lung, and the sequel is a very variable one. Occasionally sudden death occurs, but if the patient survive this initial danger all the pus may be coughed up, and complete and fairly rapid recovery follows. In other cases, however, a pyo-pneumothorax is set up, or destructive changes supervene in the lung, and the patient finally succumbs after a prolonged illness.

Another danger in connexion with an untreated empyema is the possible development, as in other cases of old-standing suppurative intrathoracic mischief, of a cerebral abscess.

Diagnosis.—To overlook an empyema is a very serious matter, as any delay in evacuating the pus exposes the patient to ever-increasing risks and renders the lung more incapable of re-expanding satisfactorily even after the pus has been let out. Pneumococcal empyemas are most commonly missed because the practitioner, having once diagnosed acute pneumonia, is apt, if the disease does not come to an end, to become obsessed with the idea of an unresolved pneumonia and to be confirmed in this opinion by misleading auscultatory signs, forgetting that by far the most likely cause of prolongation of illness after an acute pneumonia, particularly in early life, is the formation of an empyema. In streptococcal cases the constitutional symptoms are often so predominant that attention is not

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directed to the chest, and the possibility of an empyema may not be entertained; moreover, a thorough physical examination may not be at all easy owing to the patient's profound prostration.

There is one and only one reliable way of recognizing an empyema, viz. by the use of the exploring syringe whenever there is marked and persistent dullness at the base of a lung, especially after an attack of pneumonia. To rely upon any other test, especially for the distinction between a simple inflammatory and a purulent pleural effusion, is utterly fallacious. In some cases, no doubt, the constitutional symptoms resulting from an empyema are far more distinct than those due to a simple pleurisy—nobody is likely to forget this. The important point to realize is that there may be no difference between the two, and that the temperature charts of a simple and of a purulent effusion may be practically identical. It has already been pointed out that only exceptionally is there any distinction between the physical signs in the two cases, but it is perhaps not sufficiently recognized that in the diagnosis of empyema the auscultatory signs are frequently misleading rather than helpful. True to some extent of simple effusions, this is still more true of purulent ones, especially in children. The presence of bronchial or tubular breathing, with bronchophony, over the effusion often leads to disregard of the significance of the absolute dullness and increased resistance, and consequently to a diagnosis of consolidation rather than of fluid. It is true that in most cases of empyema there is a great, sometimes an enormous, increase in the leucocyte count, mainly of the polymorphonuclear cells, but there is also a large increase as a result of an attack of pneumonia, and this may persist for some weeks if resolution is delayed. If after pneumonia the patient does not progress satisfactorily and the physical signs at the base of the lung do not clear up, the absolute rule should be to explore the chest.

Exploratory puncture.—There are certain precautions which it is important to observe, in addition, of course, to the strictest antisepsis, if exploratory puncture is to yield the best results.

(a) When there is every probability from the signs present that the pleural cavity is full of fluid, it matters little where the puncture is made. Not infrequently, however, the amount of pus is very small, perhaps an ounce or even

less, and in such a case it is most important to map out the area of dullness with the utmost care, and to explore as nearly as possible in the centre of this area.

(b) It is easy to forget how very thin, comparatively, is the chest-wall of a small and probably emaciated child, and consequently to introduce the needle too far, passing it right through the pus into the lung beyond. On the other hand, some empyemas have a very thick wall, so that considerable force may be required to penetrate it.

(c) The needle should be a large one. In many cases the pus is perfectly liquid and will flow readily through the needle of a hypodermic syringe, but it may be thick and laden with flakes of lymph which may block a needle of even large bore. Therefore, if pus fails to enter the syringe the needle should afterwards be most carefully examined, as the presence of even a droplet of pus in its lumen may reveal the real nature of the disease.

(d) An empyema may be localized in an unusual or more or less inaccessible situation. Common though apical pneumonia is, it is excessively rare to meet with a localized apical empyema. Somewhat more common is an interlobar empyema, and more frequent still a diaphragmatic empyema, the pus being shut in between the base of the lung and the diaphragm. In such cases, when a first exploratory puncture fails to withdraw pus, but the history, symptoms, and perhaps also the leucocyte count point strongly to its presence, it may be desirable to make a series of punctures under general anaesthesia. An X-ray examination of the chest may help to locate the pus. It may assist also in distinguishing between an empyema and a subdiaphragmatic abscess, conditions which may be most difficult of differentiation by the ordinary methods of physical examination, and which, too, may be combined. An X-ray examination may also serve as a means of diagnosis between an empyema and an unresolved pneumonia.

It is quite exceptional for an exploratory puncture to do any harm, even if no pus is found and the lung penetrated. The writer has seen slight hæmoptysis follow, and also in one case extensive subcutaneous emphysema.

Occasionally a purulent pericarditis occurs along with an empyema and is very likely to be overlooked, but as the condition is almost invariably fatal the mistake is not of great importance.

EMPHYEMA, TREATMENT OF

Prognosis.—This depends mainly upon the following factors: (1) The nature of the causal microbe. Pneumococcal cases generally do much better than those dependent upon streptococci and staphylococci. (2) The age of the patient. The outlook is much better, as a rule, for children (except very young infants) than for adults, partly because pneumococcal empyemas are so much commoner in early life, and partly because the lung expands more readily and the soft chest-wall more easily falls in to meet a partially expanded lung in a child than in an adult with rigid chest-walls. (3) The period which elapses before an empyema is drained. Early drainage gives a better chance of recovery, partly because the longer the pus is left the more the patient's vitality is reduced and the greater the risk of complications; partly because, as pus exercises a much more injurious compression upon the lung than does a simple effusion, the lung is very likely to get bound down by firm adhesions, which may permanently prevent its re-expansion. In such cases, after drainage a large cavity is left which cannot contract, and the walls of which continue to secrete pus. Asepsis is difficult to maintain, and lardaceous disease is likely to supervene.

Treatment.—The invariable rule now is that directly an empyema is found it should receive surgical treatment. A small pneumococcal empyema occasionally dries up, especially after aspiration, but the chances of this happening are remote. In some pneumococcal infections the fluid found on exploration is turbid, but not actually purulent; it may then be withdrawn by aspiration or siphonage and the result awaited, but usually a more definitely purulent fluid reaccumulates. In cases of double empyema, which are not very uncommon in association with double pneumonia, it is advisable to open and drain one side freely at once, and to remove as much pus as possible from the other side by aspiration. The second side may be opened a very few days later, when the shock of the first operation has been recovered from. There is no danger in freely draining the two pleural cavities at the same time, as adhesions always prevent any further collapse of the lungs. (See also the next article.) J. WALTER CARR.

EMPHYEMA, TREATMENT OF.—In acute empyema the most widely practised method of treatment, until recent years, was a simple operation of rib resection and drainage. An

incision 3 in. in length is made along a rib, the periosteum is incised and separated all round (the intercostal vessels, running in a groove on the lower and internal aspect of the bone, being carefully avoided), $2\frac{1}{4}$ in. of rib is resected, the deep layer of periosteum and the parietal pleura are incised, the pus is slowly evacuated, and a drainage-tube is inserted, the wound being partially closed by sutures.

Some operators incise and drain through an intercostal space without rib resection.

The usual situations for drainage are in the midaxillary line about the level of the sixth rib, or posterior to the scapula about the ninth rib. In localized empyema the area of operation must correspond with the area of dullness. Care must be taken that, with the arm at the side, the scapula does not come to lie in close relation to the wound. A not unusual error is to place the opening too low, so that the diaphragm interferes with drainage.

There are, however, objections to these simple methods of drainage, with and without rib resection: (1) A "sucking" wound of the pleura is produced, which handicaps the mechanism of respiration and the action of the heart, and often induces collapse of the patient. (2) Collapse of the lung is produced, and its re-expansion is prevented. (3) Secondary infection, with fever and toxæmia, frequently ensues owing to the suction of bacteria from the air into the pleural cavity during the dressing of the wound. In children especially, suffering from pneumococcal empyema, the onset of acute toxæmia about the third day after operation has frequently led to disappointing results. (4) The large amount of exudate soaks the dressings profusely, and they have to be changed several times a day. (5) The collapse of the lung converts the pleural cavity with its rigid wall into a large "dead space," and a sinus is produced and heals but slowly, or it may remain open permanently, unless secondary plastic operations are carried out.

For these reasons other methods have come into use, as follows:

(1) Simple aspiration, repeated as often as the fluid reaccumulates. This is sometimes combined with irrigation of the pleural cavity through the wide-bore aspirating cannula.

(2) Incision of an intercostal space, clearing out all adherent lymph by digital manipulation and irrigation. The wound is then either closed completely, or is sewn up after the insertion of one or more rubber and glass

EMPHYEMA, TREATMENT OF

"cigarette" drains, which are removed in forty-eight hours. Fresh exudation may necessitate subsequent aspiration.

(3) An operation of valvular drainage. The writer has used this method during the last three years, and has found it give most satisfactory results.

A 2-in. incision is made over the sixth or seventh rib in the midaxillary line, or over the area of dullness if the empyema is localized. The patient lies on the unaffected side. The periosteum being incised and separated, 2 in. of rib is resected. After the incision of the pleura some of the pus is evacuated. One finger is then introduced into the pleural cavity, all septa are broken down, and the pus and masses of lymph are cleared out by irrigation with saline, introduced through a funnel and long tube; the saline is then siphoned away by lowering the funnel. A rubber tube, 1 in. in diameter and 1½ in. long, is prepared by sewing to its end a long piece of Paul's rubber tubing of 1 in. lumen. The tube is introduced so that ½ in. of it projects into the pleural cavity, and 1 in. is outside with its continuation of collapsible tubing attached. The tube is fixed in position by two strong silkworm-gut sutures passing through skin, muscles, pleura, and the sides of the tube; when these stitches are tied the wound is closed round the tube; if further sutures are needed to close the incision they are introduced through all the layers of the chest-wall. Dressings are then applied, through which the stiff rubber tube projects, and the free end of the Paul's tubing is placed in a bottle or other receptacle. In this way valvular drainage is provided, allowing the pleural effusion to escape without soiling the dressings, while the collapsible Paul's tubing prevents any fluid from being sucked back. The lung rapidly expands, and secondary infection is avoided. After about four days the lung will have become adherent to the parietal pleura, and the tube can be removed. The wound usually heals rapidly, though sometimes a shallow sinus of the chest-wall persists for a few weeks.

This method avoids all the disadvantages of non-valvular drainage and promotes early re-expansion of the lung.

If the patient is suffering from severe toxæmia, the pleural cavity should as far as possible be emptied by aspiration the day before operation, which itself should be performed under local analgesia. The skin is infiltrated with a solution of 1 per cent. novo-

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cain, and 1 c.c. of the same solution is injected in the region of the intercostal nerve accompanying the rib which is to be resected.

In cases of empyema in which the pleura is infected with several organisms, and especially in those due to gunshot wounds, Carrel's method of constant irrigation is found very useful.

In **chronic empyema** the method of valvular drainage after clearing out pus and adherent lymph is applicable to most cases, and should be combined with exercises which promote active expansion of the lungs.

If the lung is permanently collapsed and encapsulated, and there is a chronic sinus leading to a large "dead space" in the pleural cavity, one of the elaborate methods of thoracoplasty must be employed.

C. W. GORDON BRYAN.

ENCEPHALITIS.—A name applied to any non-suppurative inflammation of the brain. It covers, therefore, a number of processes which can only be differentiated by determining the agents producing them. Thus, general paralysis of the insane is a chronic encephalitis caused by the syphilitic spirochæte, and poliomyelitis is an acute inflammation of the brain produced by the virus of poliomyelitis.

It is important to remember that all forms of meningitis are associated with more or less encephalitis, and that any case of encephalitis may be complicated by meningitis. For this reason some cases of infection of the brain may be called meningitis and others encephalitis, even when the responsible organism is identical. Good examples of this are provided by the pneumococcal and influenzal infections, and it would lead to less confusion if the name "meningoencephalitis" were more generally employed.

But the position of encephalitis as a common disease among children, caused by the virus of poliomyelitis or by any of the other acute specific fevers, has not, as yet, been sufficiently recognized by the medical profession, and many cases of this disease are classed as "convulsions" or "meningitis." It may be asserted with confidence that nearly all infantile hemiplegias and diplegias, and many instances of epilepsy and mental deficiency, are the result of encephalitis in early childhood.

The brain may also become the seat of inflammation secondary to infective processes of the accessory sinuses of the skull, or as the result of injuries to the skull, and these local-

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ized inflammations may subsequently become abscesses. Septic encephalitis was a not uncommon complication of head wounds in the late war. (See HEAD INJURIES.)

Morbid anatomy.—In its essential features inflammation of the brain does not differ from inflammation of other organs. The process may be diffuse, localized, or patchy in distribution. The inflamed area is characterized macroscopically by hyperæmia, serous exudation, and often by petechial hæmorrhages giving it a congested, spotted appearance. Under the microscope the vessels are seen to be engorged with blood and their perivascular spaces filled with lymphocytic cells. Neuroglial elements are increased in number and in size. Nerve-cells and -fibres are either destroyed or may be seen in various stages of degeneration. Thrombosis of vessels with consequent necrosis of tissues may be found where the inflammation is most acute, a change which is associated with an invasion of the softened area by compound granular cells. Inflammation may be followed in favourable cases by complete anatomical resolution and restoration of function. When the nerve elements alone have been destroyed, their place is taken by neuroglial sclerosis; softened areas are transformed into scar tissue or into cysts containing straw-coloured fluid.

Symptomatology.—The symptoms of encephalitis may be described as general or local. Among the former may be reckoned headache, drowsiness, dizziness, fever, vomiting, optic neuritis, and convulsions. The mental lethargy may sometimes be replaced—for a time, at any rate—by restlessness or even maniacal excitement. Focal symptoms depend on the incidence of the inflammatory process, and are as numerous and varied as the functions of the brain. Hemiplegia, monoplegia, aphasia, hemianopia, ataxy, and cranial-nerve palsies are among the commonest disturbances of function. The symptoms and signs suggestive of meningitis, in the form of head retraction and Kernig's sign, may complete the picture. The cerebro-spinal fluid is generally liberated under increased pressure by lumbar puncture, and may contain an excess of cells during the acute stage. The causative organism may be present in the fluid, especially in cases in which the meninges are seriously involved.

Treatment consists in rest, careful nursing, and the measures usually adopted in dealing with the acute specific fevers. The administration of urotropin is generally advocated,

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but it is doubtful whether this drug really exercises any definite influence on the disease. Repeated lumbar punctures should be carried out when symptoms of increased intracranial pressure are present.

E. F. BUZZARD.

ENOEPHALITIS LETHARGIOA.—Epidemics of this disease have been recorded from time to time, but the most widespread and most serious of them visited the British Isles in 1918, and has prevailed intermittently ever since. Similar visitations have been reported in many other countries in Europe, Asia, Africa, and America.

Etiology.—This form of encephalitis differs most strikingly from others in that it attacks persons of all ages, and has no such partiality for the young as is displayed by epidemic polioencephalitis and poliomyelitis. Its seasonal incidence is also different, the majority of cases occurring in winter and spring.

Although bacteriological investigations have been made and the disease transmitted to monkeys, the responsible virus has not yet been determined with certainty.

Morbid anatomy.—This is characterized by the presence of vascular congestion, toxic degeneration of nerve-cells and neuronophagy, proliferation of the mesoblastic cells of the vessel-walls and infiltration of the nervous tissues by these cells, lymphocytic infiltration of the Virchow-Robin spaces, and glial proliferation. The changes in the blood-vessels constitute a striking feature, and the presence of small and large hæmorrhages, of areas of infarction and of thrombosis of superficial veins has been recorded in many cases.

The parts most affected by the inflammation vary in different cases, but the chief incidence of the disease appears to be on the mid-brain and brain-stem. There is reason to believe that the spinal cord is sometimes affected.

Symptomatology.—The symptoms are variable, according to the part of the brain most affected. Some cases are characterized by lethargy and cranial-nerve palsies, others give rise to the picture of paralysis agitans, and yet others are conspicuous on account of hemiplegic or diplegic phenomena. Much attention has been paid to the occurrence of involuntary movements which may affect any of the muscles of the limbs or trunk, and which may appear during the height of the disease or be delayed for weeks or months after convalescence has been established.

The onset is equally variable. The nervous

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symptoms may be preceded by a sore throat, by gastro-intestinal disorders, by hiccough, or by a localized "neuritis" of short duration but great severity. In some instances it is acute, with fever, rigors, headache, convulsions, and delirium, while in others it is insidious and drawn out over a period of days and even weeks. Not infrequently the patient complains only of slight mental lethargy and some diplopia, a condition which may last for a short period and clear up. In other cases these symptoms are followed by more serious and even fatal developments. Outside the nervous system the most striking feature has been the occasional presence of a diffuse erythematous rash resembling that of measles or of scarlet fever. Albuminuria is common at some stage of the disease, and salivation, followed by a dry mouth, may be complained of.

The mental phenomena include lethargy, which may go on to coma, as well as delirium and maniacal excitement necessitating the administration of narcotics and even physical restraint.

The cerebro-spinal fluid only presents abnormalities during the acute stage of the disease, when there may be found an excess of lymphocytes and albumin, and occasionally an increase in the quantity of glucose.

Diagnosis is often difficult. In young people lethargic encephalitis must be differentiated from tuberculous meningitis mainly by the examination of the cerebro-spinal fluid. In older patients cerebral syphilis must be eliminated by the condition of the blood and cerebro-spinal fluid, and uræmia by a careful consideration of the renal function. The simulation of cerebral hæmorrhage or cerebral thrombosis must also be borne in mind.

Prognosis.—It can only be stated that many cases completely recover, that others survive but carry with them permanent palsies or disabilities, and that an unknown percentage end fatally either in the acute stage or as the result of exhaustion and decubitus.

Treatment.—Since no specific remedy is known, treatment must be symptomatic. Urotropine may be administered in the hope that it may prove a useful ally of Nature.

In cases with profound lethargy, lumbar puncture is sometimes dangerous and rarely beneficial. Leeches applied to the head may relieve the condition, and are worth trying. Intravenous injections of salvarsan have not given good results.

E. F. BUZZARD.

ENCHONDROMA

ENCHONDROMA.—A non-malignant tumour composed of cartilage.

Multiple enchondromata of the long bones are often hereditary, and due to some abnormality in the development of the bones. Single ones are in some cases due to irregularity of the epiphyseal cartilage from rickets or old injury.

The tumour may be composed of hyalin or fibro-cartilage; it is slow-growing and has a definite fibrous capsule, from which septa proceed into the growth, dividing it into lobules. On section it appears bluish-grey and translucent; the lobules are avascular and frequently undergo fatty or myxomatous degeneration, ossification or calcification.

Varieties and treatment.—Three varieties of true enchondroma occur:—

1. In children, pedunculated enchondromata grow from the surface of long bones at the end of the diaphysis; during adolescence ossification occurs and the tumour becomes a cancellous exostosis. Symptoms are occasionally produced by pressure on nerves, and in such cases the tumour may be removed completely by operation.

2. In the metacarpus and the phalanges of the hand, and in rare cases in the corresponding bones of the foot, enchondromata, often multiple, arise in the interior of the bones and may reach a large size, destroying the peripheral bone and sometimes ulcerating through the skin; they often calcify, and occasionally they ossify. As a rule, amputation of the affected finger is necessary, but in some cases this can be avoided by enucleation of the tumour.

3. Sessile enchondromata may grow from the inner aspect of the pelvic bones and from the inner aspect of the bones of the thorax, especially the ribs near the costal cartilages. They may reach a large size and undergo myxomatous degeneration, with cyst-formation; ossification does not occur. The growths produce symptoms by compression of the structures inside the thorax or pelvis. In the pelvic variety, which is the more common, there may be severe sciatica, intestinal obstruction or, in women, interference with parturition. In the case of an enchondroma growing from the chest-wall it is often possible to remove the tumour with a portion of the rib from which it is growing; occasionally the pelvic variety can be enucleated. If removal is impossible or would be dangerous, radium should be inserted into the substance of the tumour.

ENDARTERITIS, ACUTE

ENDOCARDITIS, ACUTE

Diagnosis.—In early cases it is often only possible to settle the nature of the tumour by microscopical examination of a piece removed from its edge. *Chondro-sarcoma* is more rapidly growing and in late stages infiltrates surrounding structures. A form of pseudo-enchondroma is met with in connexion with the vocal cords and the nasal septum, due to cartilaginous proliferation as a result of chronic irritation; in the larynx hoarseness is produced, and in the nose the tumour causes obstruction.

Prognosis.—Enchondromata are slow-growing tumours, and only endanger life by their pressure effects in the chest and pelvis. In the variety which affects the long bones, growth ceases with the full development of the bone.

C. W. GORDON BRYAN.

ENDARTERITIS, ACUTE.—An acute inflammation of arteries involving all the three coats.

Etiology.—This rare disease may be caused by certain general infections, of which the principal are enteric fever, influenza, and typhus fever; rarely by puerperal infection, pneumonia, smallpox, scarlet fever, acute rheumatism, diphtheria, yellow fever, measles, gonorrhoea, and malaria. Again, it may owe its origin to general intoxications, such as gout, subacute or chronic rheumatism, syphilis, chronic lead or alcohol poisoning. Locally, an infective arteritis may occur as the result of injury and inflammation of the intima, such as may occur from an infected embolus in ulcerative endocarditis. It may also result as the sequel of clotting, or from external injury to the vessel-wall, or from surrounding inflammation.

Pathology.—The intima may be first infected by contact with the blood, or the infection may be conveyed through the vasa vasorum, when the first changes will be in the adventitia and media. Acute endarteritis has been produced experimentally by inoculating pathogenic micro-organisms and their toxins. It may be general, or localized to the great vessels and aorta. It occurs most characteristically in the arteries of the limbs, especially the lower ones. Anatomically, there occur localized thickenings of the intima associated with corresponding areas of periarteritis and mesarteritis. Microscopically, there is a cellular infiltration around the vasa vasorum.

Symptomatology.—The symptoms occur usually during convalescence from the primary

disease. They depend upon the vessels affected. In external arteries there is severe pain in the course of the vessel, provoked or exaggerated by movement, and a moderate rise of temperature, with swelling and sometimes redness over the vessel. The pain is said to be preceded by a rise in pulse-rate. Thrombosis is evidenced by a hard, painful cord, with diminution or abolition of the pulse, disturbances of sensation, and coldness and œdema of the limb. The local temperature is lowered. Gangrene is apt to follow. If there is incomplete obliteration, local elevation of temperature may occur. The arteries of the kidney, spleen, brain, and occasionally of the heart may be affected. Osler says that the condition may occur in many vessels without local disease, with high fever and signs of an acute infection.

The **prognosis** in complete obliteration is always serious.

Treatment.—If a limb is affected, rest and the application of heat are indicated. Amputation may become necessary.

OLIVER K. WILLIAMSON.

ENDEMIO HÆMATURIA (see SCHISTOSOMIASIS).

ENDEMIO NEURITIS (see BERIBERI).

ENDOCARDITIS, ACUTE.—Acute endocarditis is a result of an infection of one or more of the valves of the heart. It may be one result only of a general invasion of the heart, as in rheumatic carditis, or, again, an episode in the general invasion of many organs, when it must be co-ordinated with the other complications. If endocarditis be the only or the predominant lesion, we have still to consider the course that is usually run by the lesions produced by this particular infection; and it is apparent that endocarditis is often only an incident in the course of an infective disease.

Etiology and pathology.—The general outline of the pathology is easily understood. The infective agent gaining access to a valve produces inflammatory changes which are dependent in their nature upon the particular infection, its virulence, and the resistance of the tissues. In my opinion the infective agent is carried to the valves by the coronary circulation. The local lesion produced is termed a vegetation. The inflammation, starting in the subendothelial layer, produces tissue proliferation, cellular exudation, and necrosis. These are the elements of a vegetation which

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may or may not be capped by fibrin, and which is in any case firmly attached to the valve because it is part of its substance.

If the inflammation is acute and the bacteria are rapidly destroyed in the local lesions, there results an *acute simple endocarditis*. If it is acute but the bacteria are not overcome, there results an *acute malignant endocarditis*. All grades are found between acute simple and acute malignant endocarditis, and also between acute and chronic simple, and acute and chronic malignant endocarditis. When the acute lesions heal, the necrotic areas are replaced by scars, though not always completely. In the latter case partially healed lesions which are potential areas of danger are left behind. If the healing is complete the varying amount of thickening and distortion of the valvular segments lays the foundation of chronic valvular disease. The inflammatory processes in a valve do not always produce the familiar fringe of vegetations, but by wider dissemination in the valve structure may give rise to two other important results: a general thickening of the segments and its chords, which precedes a subsequent stenosis; or a damage to the base of the valve, impairing the resilience of its ring.

Lastly, when the endocardial lesions do not heal, but remain infested with bacteria, the general blood-stream becomes a fluid medium continuously inoculated by and in turn depositing bacteria all over the body. Sometimes this process is made more dangerous by the detachment of definite masses of bacteria in a nidus of necrotic tissue or infected fibrin, producing infarctions of various sizes. Under such conditions, again, local ulceration may eat through or destroy a segment of a valve or spread to the adjacent territories of the cardiac walls or large arteries. The occurrence of general toxæmia and of fatal cardiac injuries is easily realized as a result of such processes, which are among the gravest of all cardiac lesions.

The bacteria that may attack the cardiac valves are numerous, but those of the streptococcal group stand first. The great cause of valvular disease, rheumatism, is the result of a minute strepto-diplococcus. The majority of cases of malignant endocarditis also are the result of infections by streptococci, variously termed *S. viridans*, *S. salivarius*, Schotmüller's streptococcus, and so on. The practical point still undetermined is the relation of these micrococci to the *Diplococcus rheumaticus*. The

pneumococcus and the pyogenic streptococci are less frequent causes. The staphylococci, the gonococcus, the tubercle bacillus, the influenza bacillus, and organisms of the coli group are other recognized infecting agents. The diphtheria bacillus rarely attacks the endocardium. Cases of endocarditis that date from the common exanthemata are probably the result of various bacteria gaining access by the throat; among these the streptococci again stand first. Syphilis is usually regarded as a cause of chronic endocarditis, but the early stage may be acute in its onset and symptoms.

1. SIMPLE ENDOCARDITIS

The **clinical evidences** of endocarditis are both direct and general. Sometimes the general symptoms predominate. For example, when a violent septic infection attacks, among other organs, the heart, we may only be led to the discovery of endocarditis by the routine examination of a patient who is acutely ill. On the other hand, an acute endocarditis of slight degree but of sinister importance in the future may not cause any symptoms sufficient to call for medical advice. We have only to realize the minuteness of the lesions of such endocarditis to understand how almost by chance we may discover that a patient has an organic valvular lesion of long standing. This difficulty is one of the most important in the history of organic heart disease, and has led to the dictum that if a child suffers from signs of articular rheumatism, chorea, or repeated sore throat, the heart must be carefully examined, whether there be local symptoms or not.

Fever, some præcordial pain, palpitation, and breathlessness are frequent symptoms.

The direct evidences depend upon the valve or valves affected. There is, however, one cardinal group of signs which may be looked for—the signs of *acute dilatation*, a lesion which is often the first warning, and may precede the definite signs of an endocarditis or general carditis. Probably pallor, some fever, and possibly shortness of breath on exertion are present. The pulse rises in frequency and the tension is low. The impulse is displaced outward and is feeble; the area of deep cardiac dullness is increased to the left, and perhaps also to the right and upwards. The first sound at the impulse is short, and may be followed by a soft systolic murmur audible just internally to the left nipple and hardly, if at all, conducted outward. The second sound over the

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pulmonary area is accentuated and sometimes reduplicated. These signs may disappear, or be followed by the development of a definite *valvular lesion* which is usually that of mitral incompetence. In these circumstances the systolic murmur becomes more evident and can be traced toward the axilla, and, later, signs of hypertrophy of the left ventricle are found.

When once attention has been directed to the heart the detection of the various valvular lesions is not, as a rule, difficult. The first results will be to produce incompetence of the affected valves, for the process of stenosis is always one of gradual development.

When the aortic valve is attacked, the second sound at the aortic cartilage becomes less clear, and a systolic murmur may appear for some days before the diastolic murmur becomes audible. It must not be forgotten that in childhood this diastolic murmur is often heard more distinctly over the manubrium sterni or over the second left costal cartilage, and that at all ages, in the early stage, careful examination of the whole præcordial area may be needed for its detection. The development of a collapsing pulse may be rapid, and even, in simple endocarditis, distinct in ten days. The detection of a tricuspid systolic murmur is often difficult on account of the almost constant presence of a systolic mitral murmur; and in practice tricuspid endocarditis is seldom a serious lesion in simple endocarditis, exception being made of the rare cases of severe combined mitral and tricuspid stenosis.

Multiple valvular lesions are frequent in cardiac rheumatism. Of 250 fatal cases in childhood, while the mitral valve was damaged in all but 3, the aortic was damaged in 102, the tricuspid in 78, and the pulmonary in 6: the lesions of the last two valves were slight in degree. Special attention must be directed to the combination of aortic and mitral endocarditis producing the lesions of aortic regurgitation with mitral incompetence and often some degree of stenosis. This condition is one which, in my opinion, apart from its intrinsic gravity points to a tendency to malignancy in the rheumatic process, a statement which is supported by my analysis, made with Agassiz and Taylor, in which, of 30 cases of malignant endocarditis associated with previous rheumatism, 19 showed lesions of both these valves. The aortic valve is, as a rule, damaged at a later date than the mitral either in the same attack of endocarditis or in a subsequent one.

By the polygraph and electrocardiograph complete, but far more frequently partial, *heart-block* has been recorded in the early phase of cardiac infections, but, though such occurrences are always worthy of attention, they do not help us to any great extent so far as endocarditis is concerned. In children marked disturbances of rhythm of any duration are not usual as a feature of early endocarditis.

The great difficulties in the clinical study of endocarditis arise in ascertaining the duration and extent of its activity. Much importance is attached to a persistent *pyrexia*, together with a continuous excitement of the action of the heart, and failure in the establishment of compensation, but we are often enough in doubt as to whether it is not a carditis (see MYOCARDITIS and PERICARDITIS) rather than an endocarditis alone that is responsible. From a supervision of many cases of children with rheumatic heart disease it appears to me that we still require more data. When, in a case of acute endocarditis, the temperature remains normal, the pulse is quiet, the heart's impulse well defined and the patient gaining in weight, colour, and general strength, we feel that we are on safe ground. There are, however, many cases which are not so satisfactory. In these there are slight outbursts of pyrexia or an unduly rapid pulse, and yet we may find no sufficient explanation of the phenomena. The difficulties become greater when we are dealing with an acute endocarditis implanted upon previous organic disease, and it must be admitted that cases frequently have to leave our hospitals about which we do not feel confident; experience in a children's out-patient department has thoroughly convinced me how easily one may be deceived in these cases.

Mitral stenosis can hardly be called an acute lesion, and yet we cannot call it a chronic one. Besides the well-known stationary mitral stenosis, there is also the subacute endocarditis which precedes this phase, and in which lies the secret of the severe degrees of this lesion. We must include, therefore, in this article the signs that point to the development of mitral stenosis, looking upon the process as the result not of the healing of the minute vegetations that fringe a valve, but of a subacute form of endocarditis which for some mysterious reason is much more frequent in the female sex. Doubtless there are all grades in this process, from the acute to the subacute,

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and from vegetations fringing a cusp to the general involvement of a cusp.

When the subacute variety is developing in its purest form the valve may contract so steadily and uniformly that the signs may be most difficult to detect, and there is a stage of considerable duration in which we may be suspicious of the change but unable to decide the question. A systolic murmur, the result of the imperfect mechanism of the stiffened segments, may be audible and remain constant. On the other hand, even this may disappear. Gradually the first sound may become more abrupt, and the finger placed on the exact area of the impulse may detect a very slight thrill presystolic in time. I have heard the snap of the first sound 2 ft. from the chest-wall. Later the thrill becomes progressively more evident, and a presystolic murmur which is quite diagnostic appears and becomes longer as time passes. The second sound over the pulmonary area becomes more accentuated also. A very suspicious sign in these cases is the occurrence of tachycardia, which may be a striking feature of the early history of cases of mitral stenosis, and may remain throughout its entire course as a serious symptom. In other cases a reduplicated second sound at the impulse appears and remains constant, but this phenomenon may occur when the valve is not obviously contracted but the pericardium is adherent. The rumbling murmur may also be diastolic in time or mid-diastolic; and here again, in children, no noticeable degree of stenosis is always apparent after death.

2. MALIGNANT ENDOCARDITIS

Malignant endocarditis may occur in a previously healthy valve or in one already damaged. The most frequent antecedent at a period recent or remote is rheumatic endocarditis. To me the term malignant endocarditis conveys only this meaning—an endocarditis in which the infection gains the upper hand, with a result that is usually fatal to the patient although, exceptionally, recovery occurs, with grave damage to the heart.

It is a condition which may be very puzzling because of the severe constitutional symptoms. On this account varieties have been described, such as the pyæmic, the typhoidal, the cerebral, and the cardiac. The last provides the most classical examples, is the most frequent, and will be described first.

It may gradually emerge from an illness in-

distinguishable from acute rheumatism, or slowly develop in a patient known to have had a previous valvular disease. Again, it may flare up with great virulence in a heart previously undamaged, or in a heart already injured.

Whether ulceration or vegetative changes occur predominantly is a matter of detail dependent upon the destructive nature of the process, although the clinical symptoms may be much modified by the occurrence of one or other of them. If much resistance is offered by the tissues, vegetative processes are to be expected, and may be very extensive. In such cases the anatomical relation of these vegetations to the force of the blood-stream will lead us to expect embolic phenomena. If the local virulence is extreme, ulceration will be the great feature, the cusps of valves may be perforated or eaten away, and the chambers of the heart itself or the neighbouring large vessels eroded. If the infection is one that causes suppuration, the emboli will produce local abscesses; but if they are of the rheumatic type local necrosis will result. Both sides of the heart may be damaged, and, in comparison with simple endocarditis, valvular lesions on the right side have been said to assume a higher proportion.

Of 356 fatal cases of malignant endocarditis, 16 were examples of right-sided valvular disease alone. This means, as we should expect, that malignant endocarditis of the right side of the heart, as of the left side, when once it gains a foothold, tends to advance.

Etiology.—*Streptodiplococci* of a somewhat feeble though persistent virulence are the most important causal agents. The predisposing causes of the progressive nature of the disease are difficult to understand, though of vital interest. The probability is that damaged valves contain unhealthy necrotic, partially healed areas, and then either a fresh infection gets foothold in non-resistant tissues shut off from the protective tissue-cells, or lingering infection in these spots takes on a new and more formidable activity. A combination of infective processes may sometimes determine the result, but this is not necessary, for a single infection is able to produce malignant endocarditis.

There is some evidence in favour of the view that partially healed valves may suffer this change if the patient returns too early to work, particularly under conditions of hardship or in unfavourable surroundings. Other

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sources of infection, such as those connected with the puerperium, are also a menace to such patients. Septic throat affections and malsanitation must also be included. Whether atmospheric conditions exert an influence is difficult to decide, but there certainly seem to be outbursts of the disease. Other cases remain quite mysterious in their origin; some of these appear to me to be examples of primary malignant rheumatic endocarditis.

Clinical symptoms.—Obscure though the symptoms may be, when of the classical type no illness is more definite and easy to interpret. If it is a subacute example in a patient suffering from previous valvular disease, there is a history of loss of health of varying duration with evidence of evening fever and other signs of toxæmia, such as sweating, wasting, shivering, and sometimes definite and repeated rigors. Anæmia becomes apparent, and local signs of cardiac disturbance begin to show. A very valuable symptom is the appearance of peculiar tender points—Férol's or Osler's spots. These are usually noticed by the patient as slightly tender, raised, bluish-red areas about the size of a large pin's head on the pads of the fingers. Sometimes they are found at the wrist or in the palm of the hand, where they may be more plaque-like in shape and slightly indurated. They need inquiring for, because to the patient they appear trivial. These spots are apparently the result of minute terminal emboli, and hence their grave importance. They may precede the more definite signs of embolism, and have great diagnostic value. Clubbing of the fingers has been frequently observed in the subacute cases.

Clinical examination, in the great majority of cases, discovers active cardiac disease with persistent fever, a combination of events that is always a source of anxiety. The pulse is usually rapid, depending in character upon the particular valve affected. The heart's action is excited, and the valvular damage may suggest, either in an altered character of bruit or in the development of another valvular lesion, that the activity is located to the endocardium.

For some time no further direct evidence may be obtained, but sooner or later, in most instances, visceral emboli appear, renal, splenic, cerebral, mesenteric, arterial, as the case may be. Purpura is frequent. The spleen may be enlarged either from infarction or from a general septicæmia without infarction. In some of these cases also there may be a per-

sistent hæmaturia without infarction in the kidneys.

The temperature may run no definite course, but as a rule it gradually becomes more raised, the oscillations become wider, and the normal line is seldom or never reached. The strength fails gradually, emaciation is profound, and death may occur from sudden cardiac failure or from some accident due to embolism or ulceration of the cardiac wall. Towards the end of life diarrhœa may be a troublesome symptom, the mental faculties may become obscured, and the body-temperature, from the patient's lack of vitality, may fall below the normal line.

In the more obscure cases of malignant endocarditis the development of meningitis may divert attention to this lesion, and the *cerebral form* is thus explained. Again, although there is extensive disease, the valvular signs may be negative, and some other condition may be diagnosed, e.g. pneumonia or acute tuberculosis. In the *typhoidal form* persistent pyrexia, tremor, prostration, diarrhœa, and splenic enlargement may lead one astray. The occurrence of double optic neuritis, of petechiæ, and of emboli may clear the diagnosis, and the absence of the Widal reaction will also be of some assistance. In the *septic form* high irregular fever, sweating, rigors, purpura, jaundice, and abscess-formation all point to the severity of the process.

Some of the symptoms of malignant endocarditis may be considered a little more in detail. *Embolism* may occur in cases which do not run a malignant course, although they must always suggest a passing phase of this kind unless due to cardiac thrombus. In childhood not only may cerebral embolism produce paralysis, but it is also the most important cause of cerebral hæmorrhage. Such emboli are also the most frequent cause of aneurysm in childhood. The pain of arterial embolism may occasion grave errors in diagnosis; more than once, within my observation, it has led to confusion with sciatica and rheumatism. The *nephritis* in malignant endocarditis, apart from embolism, is a very characteristic event, and the appearance of the urine, which resembles a fluid containing laked blood, may in itself lead to the diagnosis. *Renal infarctions* may occur with no symptoms detectable during life.

A *large smooth spleen*, free from emboli, may lead us to suspect a primary blood disease, or even lardaceous disease.

ENDOCARDITIS, ACUTE

ENDOCARDITIS OF THE RIGHT SIDE OF THE HEART.—Endocarditis of the right side, though a comparatively rare event, is of considerable clinical interest. While not so rare in rheumatic disease as is believed, its slight severity, apart from severe tricuspid stenosis, makes it of small importance; but the malignant form which may have no association with rheumatism may occur in either the pulmonary or the tricuspid valve. Streptococcal infections are the most frequent, and it has been found associated with various forms of congenital heart disease.

Pulmonary incompetence.—In these cases there may be severe dyspnoea and sometimes hæmoptysis. The right side of the heart enlarges, and a soft diastolic murmur may become audible, with its maximum intensity in the left second intercostal space. This, however, is not invariable, and no murmur or only a systolic one may be detected. The absence of a water-hammer pulse assists in the exclusion of aortic regurgitation, and Gerhardt points out that the diastolic murmur becomes more audible during expiration. Hæmoptysis may be the result of pulmonary embolism or of the varying tension in the pulmonary vessels. The prognosis of these cases is necessarily grave.

Tricuspid lesions.—Only the acute and usually malignant forms will be considered. Pneumococcal, gonococcal, and virulent streptococcal infections are the most important. The chief physical signs are a systolic murmur with its maximum in the left fourth and fifth intercostal spaces close to the sternum and not audible over the back of the chest, enlargement of the right side of the heart, forcible epigastric pulsation, and a diminution in the accentuation of the pulmonary second sound. In advanced cases a ventricular venous pulse and hepatic pulsation may be present. Malignant endocarditis of this valve is rapidly fatal, and such symptoms as orthopnoea, pulmonary oedema, cyanosis, and general oedema must be expected.

Diagnosis. (a) *Simple endocarditis.*—The diagnosis of acute endocarditis of the simple form is, as a rule, not difficult if sufficient time is allowed to consider all the facts. *Functional cardiac murmurs* are sometimes most difficult to interpret, but the diagnosis of endocarditis rests on much more than the presence of a bruit. Basal systolic murmurs over the pulmonary area may be functional, or due to congenital heart disease, to some

deformity of the chest-wall, to retraction of the left lung from the region of the conus, or to mitral regurgitation from an organic lesion. In children, I know no certain criterion by which an apical functional murmur can be distinguished from an organic. I have been led to this conclusion by a study of the course of obvious mitral regurgitant lesions of rheumatic origin which have run a favourable course. At first the signs of organic disease were obvious to all who saw the cases, but gradually, as the months and years passed, the bruits were heard with more difficulty, being affected by posture and respiratory movements; doubt has then been expressed as to their nature, and, later still, more than once the heart has been declared free from disease. In adults such events as these happen less frequently.

In *simple acute dilatation of the heart* the evidence of mitral regurgitation is less definite and more evanescent, disappearing as the heart returns to the normal limits.

Congenital heart disease is sometimes almost impossible to differentiate from the acquired form, but generally, in such cases, the distinction is of no real importance, on account of the very slight degree of the lesion; or it is a question of a congenital mitral or aortic lesion in which the only point of consequence is the exact duration of the condition. Congenital and acquired lesions may coexist, and the usual clinical symptoms of congenital lesions should always be sought for in these cases of doubt. In aortic regurgitation of congenital origin the diastolic bruit may be heard at its maximum above the level of the aortic cartilage.

Slight aortic regurgitation is very easily overlooked in childhood unless careful search be made to the left of the sternum. This valve is frequently attacked in some slight degree, for, as mentioned above, of 250 fatal cases of rheumatic heart disease, it was damaged in 102 cases, about 40 per cent. The murmur of aortic regurgitation may become inaudible if there is acute cardiac failure.

Double aortic lesions may be mistaken for *pericardial friction*, and vice versa, the distinction at first being in some cases very difficult. But the character and surface depth of the sounds, the spread of friction, and the characteristics of an aortic lesion usually enable the question to be settled before long.

(b) *Malignant endocarditis.*—The diagnosis of malignant endocarditis is difficult at the

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stage when there seems to be any hope of successful treatment. The general systemic infection may entirely overshadow the cardiac signs, which in the early stages, and sometimes throughout the illness, may be very equivocal. There may be no cardiac murmur, or the presence of a slight systolic bruit may seem to be better explained as a dilatation murmur due to the fever and general illness. In these circumstances septicæmia, typhoid fever, pneumonia, or acute tuberculosis may appear the more probable explanation. The differential diagnosis clearly rests upon the discovery of cardiac lesions and, even more surely, upon finding evidence of embolism. Osler's spots, a sudden hemiplegia, a renal infarct, or a blocked artery in the extremities may clear up the difficulty. A blood-examination with the isolation of strepto-diplococci from the blood-culture may, again, lead to a right opinion. Optic neuritis may favour the diagnosis as against typhoid fever. The absence of conclusive signs of the suspected disease, be it typhoid or pneumonia, or other infection, must be allowed due weight.

Another diagnostic problem is the difficulty of deciding whether persistent fever with organic heart disease is the result of a stubborn rheumatic carditis or of the malignant form of endocarditis. The answer may only be made at the necropsy, and it is among cases of this kind that transitional examples are found, in which, so far as one can see, there has been a passing malignancy. Evidence of embolism is a valuable assistance in this diagnosis, particularly if there are clinical signs of active endocarditis in one or more valves while clear signs of a general carditis are absent. Blood-cultures are helpful, but are often negative in the early stage of the sub-acute variety, and may be positive in the severe forms of rheumatic carditis.

Yet another difficulty may arise in distinguishing between the symptoms due to emboli in various viscera, and those produced by primary disease of such viscera. In most instances of this kind the importance of the cardiac lesions which are usually present has been undervalued.

Lastly, we have to remember that there may be extensive endocarditis without the occurrence of bruits; in their absence, much significance must be attached to a persistently excited action of the heart, purpura, clubbing of the fingers, Osler's spots, and progressive anemia.

It is a good rule, if in doubt, not to hasten

to make so grave a diagnosis as that of malignant endocarditis.

Prognosis. (a) **Simple endocarditis.**—It is difficult to consider the prognosis of simple endocarditis apart from that of general carditis, for in the majority of severe rheumatic cases the myocardium and pericardium are implicated. As regards the immediate danger to life, the prognosis is invariably good. As to the future, the first point to determine is whether we have to deal with an endocarditis only, or with an endocarditis in a heart whose muscular strength has been damaged by a carditis. Having decided that the myocardium is not obviously at fault, one has next to consider the particular lesion. Slight mitral incompetence may be recovered from. A bruit may remain but the functions of the heart be unaffected. Possibly the reserve force is diminished, but there may be no evidence of this result, and the bruit in the young may disappear after some years.

On the other hand, in children there is a form of mitral regurgitation in which the valve ring has been implicated and the tone destroyed such cases run a downhill course. The heart becomes dilated and the compensation is never satisfactory. In most of these instances the myocardium is also, probably, at fault. The failure to develop proper compensation is a sufficient warning that the outlook in these cases is serious.

Mitral lesions in adult life follow the same lines, but if the attack of rheumatism occurs later in life than usual the muscle of the heart has not the same power of coping with added strain, and in all cases the habits of the patient need consideration.

The development of mitral stenosis is always a serious event, for, although there are exceptions, the majority of definite cases of this disease in hospital practice die before the forty-fifth year. The outlook is not good when symptoms of failure of compensation or embolism develop in childhood. Combined mitral and aortic lesions, broadly considered, are of gloomy prognosis. There are exceptions, for the aortic lesions may be very slight (in my opinion they may sometimes be recovered from), and the mitral lesion may also be slight in degree. These cases, however, as a rule, are associated with the severer forms of rheumatic carditis, and are very prone to develop recurrent attacks of rheumatism. If the aortic lesion predominates the condition is always serious. The majority of severe cases of this

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combined lesion die in the first twenty-five years of life either from rheumatic carditis or from malignant endocarditis.

Pure aortic regurgitation is not a common event in the rheumatic and, if the lesion is an obvious one, the prognosis must always be guarded. Some of these cases in young people are characterized by frequent attacks of anginal pain in which, while there is a large nervous element, there is also grave danger of sudden death. Aortic stenosis is essentially a chronic lesion.

In making any forecast about a rheumatic endocarditis we cannot forget, particularly in childhood, the possibility of another attack.

(b) **Malignant endocarditis.**—The prognosis depends in the first place upon the nature of the infection and the extent to which it has affected other organs. In general terms it is very serious. In a severe septicæmia the occurrence of endocarditis is usually but an event in a fatal illness.

My experience of endocarditis complicating an acute pneumococcal infection has been one of uniform gloom. Such cases have been of the malignant type, acute and rapidly fatal. Investigating a series of fatal cases of pneumonia, I did not discover a single case of early endocarditis of a simple type. The experience of other writers is different, and possibly this may be explained by climatic peculiarities. Osler records 5 cases of simple endocarditis in 100 cases of fatal pneumonia. Preble, in 132 cases, records 4 recoveries, one of them, however, a doubtful example. Among the sub-acute cases of malignant endocarditis there are examples of recovery recorded from time to time, and upon evidence which appears convincing and in accord with theoretical considerations. These exceptions do not, however, modify the general rule, and no treatment as yet promises any certain improvement.

Treatment. (a) **Simple endocarditis.**—All recent investigations point to the necessity for closely studying the means of preventing the causes of endocarditis. The condition being an infective one, the aim should be to strike at the infection itself, if possible, rather than to await its results. The indications for the prevention of the rheumatic form are considered in the article on RHEUMATISM, ACUTE.

The malignant forms should be guarded against as far as possible by insisting upon a thorough convalescence for cases of simple endocarditis and by improving the hygienic and economic conditions of patients with organic

valvular disease. Those who have suffered from any valvular disease should be particularly careful to give due time for recovery from any infective disease, such as influenza or puerperal sepsis. Chronic discharges, dental disease, and recurrent sore throat should be treated adequately. Anæmia in valvular affections should be corrected. In the poorer classes more care should be given to the choice of a suitable occupation. Though it may be thought a counsel of perfection, those who have a valvular affection should make it a rule to undergo periodical cardiac examinations, just as they make periodical visits to their dentists. Not only is it probable that we should in this way learn valuable facts about the course of endocarditis, but we should have much more chance of detecting early signs of a recrudescence and of striking in upon that phase of malignant endocarditis which is marked by a general loss of health, a phase which the patient may neglect for many valuable weeks.

The *active treatment* of endocarditis is unsatisfactory. Some believe that in the salicylates we possess a specific for rheumatic processes. (See RHEUMATISM, ACUTE.) That opinion I am unable to share, although it is clear that the value of the salicylates in painful rheumatic affections makes them also of value in endocarditis when painful symptoms are present in the joints or muscles. As to serum or vaccine treatment, or a combination of these methods, experience yields but little encouragement, although some successful results have been reported in the malignant forms (see later). No drugs, indeed, if the salicylates be excepted, appear to have any effect upon endocarditis, nor, with our knowledge of the nature of endocardial vegetations, does it seem possible that any external applications, such as blisters, can influence them in the slightest degree.

Rest is generally held to be the measure of most value, although it may not prevent the onset of endocarditis. In the acute stage it should be absolute. Its duration must be determined by the progress of the case, and not according to any arbitrary rule. There are some valuable guides upon this point, which should be used in combination. The most important of these are the temperature, the behaviour of the pulse and heart, and the patient's general condition.

If the *temperature* is obviously the result of the cardiac infection, it must reach normal

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and remain so. If the *pulse*, for the same reason, is rapid, the same rule applies. If the *heart* is dilated, it must return to the natural size or, failing this, must develop clear signs of hypertrophy. These results obtained and assured, the treatment may be modified by degrees, the same tests being applied as each forward step is taken. The steps are (1) an extra pillow; (2) sitting up in bed; (3) sitting up on a couch and chair with feet up, and then with feet down; (4) walking along the flat and up slight inclines; (5) exercise, gradually increasing in variety and vigour.

For those who can afford it, skilled massage while in bed is a useful adjunct.

The *general condition* of the patient should show a gain in weight and general cheerfulness. Each of the indications that have been considered is of value, but we have to bear in mind that a mysterious fever may be present which is dependent not upon the endocarditis but upon some other manifestation; the pulse, again, may be quick from nervous influences and not from endocarditis. It is therefore a combination of the indications that forms so useful a guide, for even if a premature forward step has been made it can be corrected before harm is done.

In childhood absolute rest should not be maintained a moment longer than is necessary, for children respond well to prudent forward efforts which bring to them change and hope. With adults more caution is needed. In all cases there is much advantage to be found in a regular programme, by means of which we are the better able to give time for the vegetations to be securely healed by scarring, for the cardiac wall, which is invariably weakened by the infective process, to get firm, and, when there is decided damage to the efficiency of the valve, for hypertrophy and compensation to be established.

It is very inadvisable to let either children or adults hear too much about their cardiac lesions, for there is no organ over which anxiety and the imagination can exert a more harmful influence.

During the periods of complete rest and convalescence various medicines may be indicated. The digestion and bowels must be carefully supervised. *Digitalis* is of some service, and particularly when active exertion is being attempted. Opium may be needed to soothe the nervous system, and bromide for a nervous and excitable pulse and cardiac action. Other indications will suggest them-

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selves. The *diet* should be light and digestible, but there is no need to exclude meat and soups in the rheumatic cases when convalescence is clearly established. I can see no association between diet and rheumatism in childhood.

(b) **Malignant endocarditis.**—The treatment of malignant endocarditis is on the same general lines as that of simple, and often enough the measures employed are palliative and suggested by the emergencies that arise. It is to specific therapy that we turn for help, but my experience has not been encouraging, and failures have been repeated. I am indebted to Dr. Teale, Lecturer on Bacteriology at University College Hospital, for a synopsis of his views upon these methods. He holds that antisera are of particular value when the degree of general toxæmia is high, for by their use already-formed antibodies are brought into action. Their value is nevertheless limited by dilution and by the short duration of their action. He advocates one large dose where the toxæmia is high, and couples with it the employment of a vaccine. In order to save time the first injection may be from a stock vaccine, and should always be small in case active changes should occur in the vegetations and emboli result. The further use of vaccines, which should be autogenous, will be guided by a study of the temperature, pulse, general condition, and urine. The indications for the next dose will be a rising temperature, and the increase in the dosage should be cautious. If possible, the antiserum should not be repeated unless there is grave toxæmia. The vaccine may need to be repeated every third or fourth day, in accordance with the rules given above. Dr. Teale is of opinion that sensitized vaccines offer no advantage in this condition.

F. J. POYNTON.

ENDOCERVICITIS (see ENDOMETRITIS).

ENDOMETRITIS, ENDOCERVICITIS, METRITIS.—These closely related subjects will be considered in the order in which they are named.

ENDOMETRITIS

This term is often used rather loosely because it is sometimes difficult to determine whether certain changes in the endometrium are due to inflammation or not. Hypertrophy and hyperplasia of the glands occur in the normal endometrium in the changes associated with menstruation. Formerly these changes were regarded as inflammatory, but they are

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now known to be physiological. The same remark applies to the atrophy that occurs after the menopause.

Adenomatous overgrowth is a frequent cause of thickening of the endometrium. No evidence of inflammation can be detected microscopically in this condition, and as it is by no means uncommon in virgins the clinical evidence points to its non-inflammatory origin. This form of thickening of the endometrium must be regarded as a new growth.

Etiology.—The cause of endometritis is infection, but in a large proportion of chronic cases it is not possible to determine the nature or even prove the existence of the organisms.

Gonorrhœal and puerperal infection are the commonest and most important causes of endometritis, and in these instances the clinical and microscopical evidence of infection is often complete. But as it is impossible to get cultures from the interior of the uterus without the risk of contamination from the cervix, bacteriological proof is extremely difficult, except in the rare cases in which the uterus is removed in an early stage of the disease.

Other organisms also undoubtedly invade the endometrium from the vagina, and the tubercle and colon bacilli from above by way of the Fallopian tubes.

Pathology.—The microscopical recognition of inflammation in the endometrium is much more difficult than in the case of most other tissues of the body, because only curettings can be obtained, as a rule, for examination. The endometrium, too, during menstrual life is constantly changing in preparation for, or in recovery from, a menstrual period, and in this process its stroma becomes oedematous; there are other features also that resemble the changes caused by inflammation. For details the reader must be referred to works on pathology, but it may be stated briefly that a conclusion is reached mainly on such points as the relative abundance or scarcity of polymorphonuclear leucocytes and plasma cells in the stroma.

The interstitial tissue of the endometrium is the part primarily affected by inflammation, and two well-marked varieties of endometritis are recognized, the atrophic and the hyperplastic. In the former the endometrium becomes thinned and the glands are reduced in size by pressure exerted on them by the dense stroma; in the latter there is general hyperplasia of the glandular elements as well as of the stroma, which becomes dense and fibrous.

After the menopause the atrophied endometrium frequently becomes infected by pyogenic organisms, and a similar condition occurs in cases of pyometra before the menopause when the cervix has been occluded by a malignant growth. In these cases the surface epithelium is shed and the endometrium shows pronounced evidence of inflammation.

Symptoms.—When *acute* endometritis is due to puerperal septicæmia and sapræmia, its most frequent causes, the symptoms characteristic of these conditions will be present.

Gonorrhœa often spreads rapidly from the vagina and cervix to the endometrium, and next to puerperal infection is the commonest and most important cause.

Other organisms may spread from the vagina, and the presence of a necrotic fibroid or a breaking-down carcinoma or the introduction of a dirty instrument into the uterus may cause acute inflammation. Increase of vaginal discharge is to be expected when the endometrium becomes inflamed, but often the existence of the condition is uncertain or unsuspected until the pelvic peritoneum becomes involved by a further spread of the infection.

Chronic endometritis is a minor complaint, which frequently has existed for years before medical advice is sought. Leucorrhœa, menorrhagia and sometimes metrorrhagia, dysmenorrhœa, and pelvic pain are its characteristic symptoms. A patient will often complain of these symptoms dating from a labour or miscarriage or an acute attack of vaginitis. There may have been no disturbance obvious to her during the puerperium, nor any acute illness following the vaginitis. The leucorrhœal discharge is often very profuse, and may come largely from the cervical mucous membrane, which is almost inevitably infected in cases of endometritis in which the infection has come from the vagina; there is, however, no means of proving the actual source of the discharge. The uterine bleeding is not usually severe, but the periods are prolonged and the loss increased. Irregular losses between the periods for a day or so may occur, and in exceptional cases coitus provokes slight bleeding, which may persist for twenty-four to twenty-six hours. The dysmenorrhœa is usually of the congestive type, but spasmodic attacks may occur. There is often a feeling of weight and discomfort in the pelvis or definite bearing-down between the periods.

Sterility is frequently due to endometritis, and patients often seek advice on account of this symptom only.

ENDOMETRITIS, ENDOCERVICITIS, METRITIS

In cases of senile endometritis foul blood-stained discharge is the symptom that brings patients for treatment.

In some cases of endometritis the general health becomes impaired; there may be headache, backache, and vague pains in other parts of the body, and women often complain of feeling weak and incapable of any physical exertion without being unduly tired.

Diagnosis.—Bimanual examination may elicit tenderness of the uterus, which is bulky and its cavity slightly elongated. The cervical mucous membrane and Fallopian tubes are also very frequently infected in endometritis, so that some collateral evidence may be obtained by finding thickened tubes, erosion, mucous polypi, or some other sign of old pelvic inflammation.

The clinical features are rather indefinite, and the diagnosis ultimately rests on the microscopical examination of the curettings; when, however, there is a history of infection with the above symptoms and physical signs the probabilities are that the case is one of endometritis.

Prognosis.—The results of dilatation and curetting are good, and a cure by this operation can be expected in the great majority of cases. In some the full benefit may not be obtained for several months, although ultimately it becomes complete; occasionally it is necessary to repeat the operation.

Treatment.—Mild cases may require very little treatment, and often douches are enough to relieve the patient of the discomfort of the leucorrhœa and to reduce the excessive loss of blood. When symptoms are troublesome, curetting, followed by swabbing of the uterus with strong iodine, should be undertaken. If the general health is poor a change and rest, with appropriate medical treatment, should be advised after the operation.

ENDOCERVICITIS

Inflammation of the cervical mucous membrane is caused by infection from below. Gonorrhœa is much the most important of such infections, and frequently attacks the cervix primarily.

Other infecting agents are the streptococcus and numerous other organisms; after an attack of gonorrhœa it is common for the damaged cervical mucous membrane to be invaded by these organisms, which keep up the inflammation long after the gonococcus has disappeared.

When the cervix is lacerated and the lips

become everted, the exposed mucous membrane readily becomes infected and inflamed.

Pathology.—The cervical mucous membrane is red and swollen, and polypi develop on it. The cervix itself is often enlarged, and areas of erosion form around the os. Small shotty cysts containing mucoid fluid, known as Naboth's follicles, are commonly seen on the surface of the cervix, and are caused by retention of the secretion in the glands. Microscopically, round-celled infiltration and fibrosis of the stroma are found in the mucous membrane.

Symptoms.—Leucorrhœa is always present, the discharge varying greatly in quantity and character. It may be thick, purulent or blood-stained, or watery in consistence, but that coming from the cervix is commonly clear and viscid like the unboiled white of egg. Backache is common. Frequently there are symptoms which point to a spread of the inflammation to the endometrium and Fallopian tubes.

Diagnosis.—The presence of leucorrhœa, the appearance of the cervix, and the history of the case make the diagnosis clear, but it is impossible to say whether the inflammation is limited to the cervix or has spread to the endometrium.

Treatment.—Douches of alum and zinc sulphate, 30 gr. of each to a pint of water, or some mild antiseptic often give good results, and should first be tried. If the progress is not satisfactory the cervical canal and exposed glandular mucous membrane should be swabbed in addition three or four times at weekly intervals with strong iodine, pure carbolic, silver nitrate 10 per cent., or one of the organic silver preparations. This treatment often has a good effect, but if it fails a course of ten days or a fortnight of medicated pessaries containing ichthyol 5–10 per cent., glycerin 50 per cent., or iodine $\frac{1}{4}$ –1 per cent. should have a trial in place of the swabbing.

If the discharge still persists, the cervix should be curetted and swabbed, and, as there may be endometritis too, the interior of the uterus should be treated in the same way.

Good results have been reported by Hobbs (*Pract.*, Jan., 1921) in the early stages of gonorrhœa by swabbing the cervical canal with tincture of iodine 1 part and glycerin 8 parts, and in chronic cases of endocervicitis by introducing gauze tampons saturated with liniment of iodine 3 parts and glycerin 1 part into the cervical canal. When this treatment fails to

cure the discharge, he advocates dilatation of the cervix and irrigation of the cavity of the uterus with liniment of iodine, followed by the introduction for six hours of an intra-uterine tampon soaked in equal parts of tincture of iodine and glycerin.

In some cases of eversion of the lips, repair of the cervix gives good results, while in others, with lacerations or extensive erosions, amputation of the cervix is the most satisfactory treatment.

METRITIS

Inflammation of the muscular wall of the uterus may be acute or chronic, and occurs as a sequel of endometritis.

Acute metritis is the result of puerperal infection in the great majority of cases. When a piece of infected placenta or membrane is retained in the uterus an inflammatory zone deep to it develops in the uterine wall. A less well-defined layer is also found under the endometrium in cases of puerperal septicæmia.

If the infection is virulent the vessels become thrombosed, inflammatory foci form near them, and abscesses may develop in the uterine muscle.

Chronic metritis is a term used to indicate a clinical rather than a definite pathological condition. These cases of "chronic" metritis have been classified by Fletcher Shaw under three headings:—

1. True metritis, which is characterized by the development of an excess of fibrous tissue in the wall and accounts for 1 per cent. of all cases.

2. Subinvolution, in which there is excess of elastic and fibrous tissue; this group constitutes 95 per cent.

3. True hypertrophy, in which the various elements of the uterine wall are all increased; this provides the remaining 4 per cent.

Etiology.—It will be seen from the above that inflammation is only definitely responsible for about 1 per cent. of the cases, whilst subinvolution, which is very probably itself due to a mild infection during the puerperium, accounts for the great bulk. The cause of the hypertrophy is unknown.

Pathology.—The wall of the uterus is tough and often double its normal thickness; its cut surface is pale and fibrous in appearance, and the divided vessels stand out very obviously upon it. The whole organ is uniformly enlarged, the cavity sharing in the enlargement. The endometrium may be thickened or abnormally thin.

Symptoms.—The chief and frequently the only symptom is profuse and increasing bleeding, but there may be leucorrhœa also. At first the bleeding is definitely menstrual in character, but the periods increase in duration and the loss may become practically continuous.

Chronic metritis is usually seen in women who have had children and are approaching the menopause.

Diagnosis.—The uterus is bulky and harder than normal, and the cavity is found by the sound to be slightly elongated.

Until the cervix is dilated and the cavity explored with the finger no definite diagnosis of chronic metritis can be made, as it is impossible to be certain that the signs and symptoms are not due to a small submucous fibroid. But with the finger in the uterus the diagnosis can be established, for the thickening of the wall can be recognized and the presence of any tumour in the wall or gross change in the endometrium excluded.

Treatment.—Drugs have practically no influence on this type of uterine bleeding, but are usually given a trial because the cause cannot be determined with certainty.

If the patient has already become very anæmic, delay in stopping the bleeding may be dangerous; in such cases curetting and exploration of the uterus should be done without delay. In this way the cause of the bleeding will be ascertained, and even if it is due to metritis there is some hope that the loss will be reduced.

When the endometrium is found to be thickened, curetting often gives temporary relief; but if it is thin, very little can be expected from the operation.

If, after curetting, the symptoms are not relieved, the usual and probably the best course is to remove the uterus. The alternative is treatment by X-rays or radium; the latter seems the more satisfactory, as its effect is probably more rapid. A tube containing 100 mg. of radium can be introduced into the uterus at the time of the curetting and left in position for twenty-four or thirty-six hours. After its removal there may be complete cessation of the bleeding.

J. P. HEDLEY.

ENEMATA.—Fluids are injected into the bowel to subserve two purposes, the one to supply nutriment when for some reason it is either impossible or unwise to give food in the ordinary way, the other therapeutic.

ENEMATA

Nutrient enemata.—These were formerly administered much more frequently than at present. Examples are :

Peptonized milk	3ii.-iv.
Beef tea	3ii.
Raw-meat juice	3i.
Sod. bicarb.	gr. x.
Liq. pancreat.	3i.
Beef tea	3ss.
Solution of glucose (80 per cent.)	3ss.
Sod. bicarb.	gr. x.
Liq. pancreat.	3i.
Milk	3iii.

Much doubt has been thrown on the value of giving proteins rectally, since in nutrient enemata, as usually administered, they are provided in the form of peptones, and physiology now teaches that they can only be absorbed as amino-acids. Careful observations on patients have borne this out and have also shown that the fat of milk is absorbed in very small quantity. Moreover, after these enemata the residue washed out from the bowel is very offensive, and if they are continued for several days the patient often manifests the symptoms of putrefactive intoxication. The difficulty may be surmounted by giving chemically prepared amino-acids, or milk which has been pancreatized for twenty-four hours so that the peptones first formed have been converted into amino-acids.

The best kind of food for rectal alimentation is undoubtedly glucose, a 6-per-cent. solution of which in tap-water is isotonic with blood; half a pint to a pint may be given every four hours. The glucose may be combined with milk pancreatized for twenty-four hours. With all forms of rectal feeding the bowel should be washed out with warm saline at least once a day, and thirst must be combated by leaving in it from half a pint to a pint of the solution. A further half-pint or pint of saline during the twenty-four hours is advisable. Great care must also be paid to the cleanliness of the mouth.

Medicinal enemata may be divided into those which act locally and those which are used as a means of administering a drug the general action of which is desired.

Among enemata employed for their local effect are :

1. **Purgative enemata.**—These are usually large ($\frac{1}{2}$ –1 pint or more according to age), and may be of warm water only or contain a

slightly irritating substance such as soap. When soap is added it is advisable to use the soft soap of the British Pharmacopoeia (1 oz. to the pint of water), lest enemata rashes or other signs of toxæmia follow. The result in either case is to provoke an active peristalsis. A similar effect may be attained by the use of smaller quantities of more irritating substances, for example glycerin (1 oz.). This variety of enema is useful when distension of the colon must be avoided, but is too irritating to be employed frequently. When the bowel is filled with hard, dry scybala, enemata of warm olive oil ($\frac{1}{2}$ pint), to which castor oil (1 oz.) may be added, given very slowly and retained for as long as possible, are sometimes effective after other measures have failed. Purgative enemata should not be employed for constipation which can be treated successfully by other measures, and should be discontinued as soon as possible, since, by distension, they are prone to weaken an already atonic bowel, and if continued too long may be necessary for the remainder of the patient's life.

2. **For tympanites.**—A simple purgative enema may suffice, especially if a two-way tube is used, but when the tympanites is severe, particularly in the absence of scybala, a turpentine enema ($\frac{1}{2}$ oz. added to 10 oz. of mucilage of starch) or spirit. menth. pip. (1 oz. to a pint of water) is more satisfactory.

3. **Antiseptic and astringent enemata** are employed for local affections of the colon—for example, ulceration from dysentery or non-dysenteric colitis and stercoral ulceration. Among the solutions employed are listerin, glycothymolin, borolyptol, or boric acid (1–2 oz. to the quart), acetozone (1 to 1,000), protargol and argyrol (1 to 500), quin. sulph. (1 to 500–1,000), silver nitrate (1 to 2,000), thymol (1 to 2,500), and pot. permang. (1 to 2,000).

4. **To control hæmorrhage and diarrhoea** a starch-and-opium enema (tinct. opii 15 min., mucil. amyli to 2 oz.) is commonly used and is often efficacious. Such treatment is not strictly local, for the opium has no direct astringent effect. For the bleeding from colonic ulcers, injections of gelatin (10 per cent.), or gelatin (5 per cent.) containing calcium chloride (2 per cent.), are often more beneficial than opium and starch. Ice-water, or water at as high a temperature as can be borne, sometimes controls persistent and intractable bleeding.

5. **For tenesmus** a small simple enema of

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warm water often gives immediate relief by clearing out irritating scybala and promoting relaxation of the spasm of the sphincter.

Among medicinal enemata employed for their **general** effect—(1) large *saline* enemata are used to combat shock, to replace loss of body fluids, to dilute circulating toxins as in uræmia, and to allay thirst. The injection of saline continuously by the bowel after surgical operations, especially abdominal operations, has proved of the greatest value.

(2) Drugs are administered in this way when they cannot be given orally, as in mental disease, tetanus, or when the patient is unconscious or convulsive; examples are chloral and bromides in epilepsy, and quinine in cerebral malaria.

(3) The colon is utilized as an accessory channel of absorption when large quantities of a drug are indicated. Thus, in the diacetic-acid poisoning of diabetes, alkalis and lævulose are administered rectally at the same time as orally, and the same is true of glucose and alkalis in delayed anæsthetic poisoning and cyclical vomiting.

(4) General anæsthesia is produced by the rectal injection of a solution of ether in oil, a form of administration specially indicated when it is desired to render the upper part of the body anæsthetic while allowing free access to the mouth and face, as in plastic and cosmetic operations. **FREDERICK LANGMEAD.**

ENOPHTHALMOS (*see* EYE, EXAMINATION OF; ORBIT, AFFECTIONS OF).

ENTERIC FEVER (*see* TYPHOID AND PARATYPHOID FEVERS).

ENTERITIS.—The acute catarrhal forms are described under **DIARRHŒA** and **DIARRHŒAL DISORDERS OF INFANTS**; other varieties are considered in connexion with the conditions in which they occur.

ENTEROPTOSIS (*see* VISCEROPTOSIS).

ENTEROSPASM (*see* CONSTIPATION).

ENTROPION (*see* EYELIDS, AFFECTIONS OF).

ENURESIS (*see* INCONTINENCE OF URINE).

ENZYMES (*see* PATHOLOGY, CHEMICAL).

EOSINOPHILIA (*see* LEUCOCYTOSIS).

EPHELIS AB IGNE (*see* ECZEMA).

EPIDEMIC CEREBRO-SPINAL FEVER (*see* MENINGITIS).

EPIDIDYMITIS

EPIDEMIC DIARRHŒA (*see* **DIARRHŒAL DISORDERS OF INFANTS**).

EPIDEMIC HÆMOGLOBINURIA (*see* Epidemic Jaundice of Infants, under **JAUNDICE**).

EPIDEMIC JAUNDICE (*see* **JAUNDICE**).

EPIDEMIC PAROTITIS (*see* **MUMPS**).

EPIDERMOLYSIS BULLOSA (*see* **PEMPHIGUS**).

EPIDIDYMITIS.—Inflammation of the epididymis. As the inflammation commonly extends to the body of the testicle, the term *epididymo-orchitis* is often employed.

Etiology.—Except when due to injury, epididymitis is always secondary to an infection of the posterior, or prostatic, part of the urethra. Frequently some irritation or slight injury, such as may be caused by the passage of catheters, is a contributory and exciting cause. The usual source of infection is gonorrhœa, the epididymo-orchitis appearing about the fourth week of the discharge, though it may occur much later, in the course of a chronic gleet. In some cases it appears to be brought on by the use of strong injections. It may also occur with gouty urethritis and in cases of stricture and enlarged prostate, especially when catheters have to be employed. Vesical calculus, a small calculus impacted in the urethra, and prostatic calculi may also provide the source of infection and irritation; and here again the use of instruments, such as a lithotrite, may be the actual exciting cause. Lastly, epididymo-orchitis may occur with a *Bacillus coli* infection of the bladder and prostate without urethral discharge, and tuberculous disease may have such an acute onset as to suggest gonococcal infection.

The general view that the inflammatory process is a direct extension along the vas, and possibly also the lymphatics of the cord, is confirmed by the thickening and tenderness of this structure. The globus minor is first involved, the inflammation subsequently extending to the globus major and the testicle. While the vas and the epididymis contain pus and shed epithelium, the testis usually shows only œdema and congestion. A small acute hydrocele is commonly present.

Symptomatology.—At first there is pain and aching in the groin and iliac region, which subsequently extends to the testicle. The globus minor first enlarges, but the swelling

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rapidly extends to the whole of the epididymis, so that it appears to wrap round the body of the testis, which sometimes cannot be separately distinguished. The swelling is extremely tender, and there is a characteristic severe sickening pain. Constitutional disturbance may be severe, as shown by pyrexia, vomiting, and occasionally by rigors. There is redness of the skin with cedema of the scrotal tissues, and, as the result of the latter, the swelling is flattened laterally owing to the pressure of the thighs. The inflammation reaches its maximum in five or six days, and then, after three or four days, subsides, at first slowly. The acute symptoms follow the same course, but some swelling of the globus minor persists for a long time, in some cases almost indefinitely. It is rare for both testicles to be affected simultaneously, but as one improves the other may become inflamed. Abscess-formation is very rare in gonorrhœal cases, but suppuration in the tunica vaginalis, cord, or epididymis may occur with other forms, especially after operation, or there may be actual sloughing of the testicle. In cases in which there is persistent thickening of the globus minor the lumen may be obliterated, and, if this is bilateral, sterility will result.

The urethral discharge commonly disappears as the epididymitis develops, but reappears as the inflammation subsides.

Diagnosis.—Epididymitis must be distinguished from orchitis, which is due to entirely different causes. Tuberculous disease with an acute onset may simulate acute epididymitis, but abscess-formation and bacteriological examination of any discharge which may be present will clear up the diagnosis. The persistent thickening of the epididymis may also be mistaken for tuberculous trouble. When occurring in an imperfectly descended testicle in the inguinal canal, it may closely resemble strangulated hernia, or, if the testicle is intra-abdominal, peritonitis or appendicitis.

Treatment.—The patient should be kept in bed with the scrotum raised. In the early stages cold, in the form of an ice-bag or an ice poultice, gives the greatest relief, but after forty-eight hours hot lead-and-opium fomentations are more effective. The bowels should be kept acting freely with calomel followed by saline purges. Morphina may be required in the acute stage. After the acute symptoms have subsided, glycerin and belladonna may be applied locally, and later, when the pain has disappeared, strapping over Scott's dressing

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promotes the absorption of the swelling. Potassium iodide should also be given in the late stages. When the inflammation has diminished, the urethritis must be treated, and in non-venereal cases the cause of the infection and irritation in the urethra must receive attention.

Should suppuration occur, the abscess must be opened and drained.

For tuberculous epididymitis, see TESTIS, TUBERCULOSIS OF.

PHILIP TURNER.

EPILEPSY.—A sudden loss of consciousness must be regarded as the most remarkable and characteristic feature of epilepsy, and one which has up to the present time lacked a satisfactory explanation. If it were understood, it would not be difficult to explain the warning which sometimes precedes it, or the convulsive seizures and mental disturbances which follow it. That anyone may suddenly lose consciousness and present all the features of an epileptic attack, provided the provocation is sufficient, can hardly be disputed, but, for practical purposes, persons who display this tendency without obvious and sufficient cause are regarded as epileptic, and as suffering from a disease of which the outward and visible sign is the epileptic seizure.

Looked at from the experimental point of view, we know that sudden loss of consciousness, followed by convulsive movements, can be produced by a failure of circulation in the brain, and in seeking for an adequate cause of it in an epileptic attack it is difficult or impossible to conceive that it can occur except as the result of a sudden interference with the circulation in the higher nerve-centres. The cessation of the heart's action which precedes some epileptic attacks, and the epileptiform seizures which are associated with heart-block, support this view. But the sudden loss of consciousness may occur without evidence of cardiac failure; this suggests some vasomotor disturbance confined to the cerebrum, producing a transient ischæmia of important centres.

Etiology.—The constant factor is, without doubt, an inherent instability, the exact nature of which is not yet understood. An investigation of the family history of epileptics reveals, in the majority of instances, a predisposition to epilepsy, insanity, mental deficiency, psychoneurosis, a drug habit, or alcoholism. It is tempting to assume that the inherent instability, in the case of the epileptic

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patient, involves the vaso-motor system, and this view finds some support in the frequent association of vaso-motor disturbances in the limbs with epilepsy. Physical defects of other kinds, generally regarded as degenerative stigmata, are frequently present.

Age.—The first manifestations of epilepsy are most common during infancy, during the second dentition, or about the age of puberty. A history of convulsions in infancy must be looked upon with some suspicion, as they are not infrequently provoked by an unrecognized encephalitis, which may be responsible for some permanent structural defect of the brain that forms a starting-point for later seizures.

In quite a large proportion of epileptic patients the first fit occurs during the unstable period of puberty, and cases are by no means uncommon in which a few fits at that age are the sole manifestation of the epileptic diathesis. True epilepsy does not often commence after 25 years of age; when this occurs it is usually the result of exposure to some severe mental or physical strain, to the effects of alcohol, lead, or syphilis, or to senile degenerative changes.

Reflex irritation.—It is difficult to apportion the responsibility of such factors as dentition, worms, errors of refraction, or nasal defects in the production of epilepsy, although it appears probable that they may be reckoned as exciting causes in a certain number of cases. Blows on the head, trivial or severe, are not uncommonly followed by a first epileptic attack, but they cannot be regarded as more than secondary factors. Injuries to the brain at birth must be included among possible causes.

Psychical influences.—The part played by mental disturbances in the production of epileptic attacks has received considerable attention in recent years, and there can be little doubt that fear, disappointment, and emotional excitement are important factors in provoking the first manifestations of the disease, and may play a part in its subsequent development.

Morbid anatomy.—The examination of epileptic brains has revealed no constant features which can be regarded as specific. Moreover, it is almost impossible to discriminate between the results and the causes of the disease. Degeneration of neurones and hyperglia in longstanding cases may fairly be regarded as the consequences of an epileptic life. The presence of congenital defects, scars, porencephaly and other evidences of preceding

inflammation or traumata, throws no light on the pathology of epilepsy, although it may explain the aura and clinical characters of the patient's seizures.

Symptomatology.—An epileptic seizure may be preceded by a prodromal period, varying in length from a few hours to a few days, during which the patient is depressed, irritable, and restless, or perhaps unduly elated and energetic. But these prodromal states are not very common. The sudden or rapid loss of consciousness which is the essential feature of an epileptic attack may last for a fraction of a second only or for a much longer period. In the former case it may be the only manifestation of an attack, and may be so transient that it escapes the notice of the patient and of his companions. Such attacks may recur for months or years without being recognized, till the incidence of convulsions draws attention to the more frequent *petit mal*.

The aura.—In a large proportion of epileptics loss of consciousness is immediately preceded by an aura, which may be sensory, motor, or psychical. The aura in each individual is nearly always the same, and it may be therefore an indication of the locality in which the cerebral disturbance commences. An epileptic may state that some of his fits only are ushered in by an aura, but this may be due to failure to remember the warning, or to a retrograde amnesia after the more severe attacks.

A *sensory* aura may consist of a visceral sensation referred to the stomach or the chest, or paræsthesiæ or pains in the limbs, or it may be a special sense phenomenon, as flashes of light, musical sounds, or peculiar tastes or smells. A *motor* aura may consist of local twitchings or spasms confined at first to a few muscles, but showing a tendency to spread along a definite line of march. More purposive movements, such as locomotion, must be regarded as the emotional expression of a psychical aura. *Psychical* auras are not uncommon; the patient may complain of dreaminess, or of some emotional or intellectual disturbance, which may or may not be manifest in his behaviour. Finally, it must be remembered that an aura may occur without subsequent loss of consciousness. Not infrequently it is the only manifestation of an epileptic state.

Loss of consciousness.—The sudden loss of consciousness may be so short-lived that the patient maintains his position, sitting or standing; more often it is the cause of his headlong

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fall to the ground. The length of unconsciousness is often difficult to measure, as the patient may pass from it into a deep sleep.

Convulsive movements.—These are at first tonic in character, all the voluntary muscles being involved in spasm more or less simultaneously, or those on one side of the body perhaps slightly before those on the other. They are often accompanied by a loud cry, probably produced by a forced expulsion of air through a narrowed outlet. The head is usually retracted and turned to one side or the other; the eyes may be open and staring, the jaws clenched, the chest fixed, and the whole trunk in a position of over-extension. The arms may be extended, but are more usually flexed at the elbows and wrists, with the fingers and thumbs strongly adducted and clenched. The legs are extended, with the feet and toes pointing inwards and downwards. During this tonic phase, which may last from a few seconds to half a minute, the patient's complexion becomes livid and his lips are cyanotic, while the pupils dilate and are inactive to light.

The tonic phase passes imperceptibly into one of clonic spasm: twitching of the eyes, the face, or the limbs on one side gradually increases in frequency and spreads until the whole body is involved in violent convulsions. Masticatory spasms are responsible for biting of the tongue during this stage, and a frothy saliva, which may be bloodstained, is churned from the mouth. At the same time the urine and feces may be evacuated. Cyanosis increases, the pulse becomes more rapid, and the skin is covered with cold sweat. The convulsions subside gradually, and two or three minutes may elapse before they disappear. If the movements are very violent, injuries such as fractures and dislocations may result.

The above description is that of a severe attack, but many epileptic seizures may be associated with spasms which are hardly noticeable or may be limited to a few muscles of the face, the eyes, or the limbs.

As the spasms pass off, the patient relaxes into a condition of flaccidity associated with deep respiration, a gradual diminution in the size of the pupils, and return of consciousness, which, however, may be masked by heavy sleep. Examination of the reflexes at this stage may reveal an exaggeration of the tendon-jerks, absence of abdominal reflexes, and extensor responses to plantar stimulation.

A petit mal has all the features of a grand

mal except the convulsive stage and the subsequent exhaustion.

Post-paroxysmal phenomena.—There is often a period of automatism associated with mental confusion after both major and minor attacks. The patient may dress, undress, carry out purposeless acts, or inflict injuries on himself and others. Some patients may remain for hours and even for days in a condition of mental excitement and restlessness associated with delusions. Less commonly the paroxysm leaves a condition of hemiplegic weakness, or true epileptic attacks may be followed by purely hysterical phenomena. The post-paroxysmal symptoms are important from the medico-legal point of view owing to the commission of criminal offences, and it is necessary to remember that they are as likely to follow an attack which is so slight as to escape recognition, as one which has all the characteristics of an epileptic seizure. Most patients, however, make a complete and rapid recovery from an epileptic attack, and are able within an hour or two to resume their ordinary occupations, but some are exhausted for considerable periods and others suffer with vomiting and headache.

Frequency and time-incidence of attacks.—Frequency varies from many attacks a day to one a month, one in three months, one in a year, or even longer intervals. In many cases there is a serial tendency, three or four attacks in a day, or fifty to a hundred attacks in the course of a few days, being followed by a long period of immunity. Some regularity in the incidence of attacks is not uncommon; they may have definite association with the menstrual period in the case of women. Attacks may be entirely nocturnal or may occur only during the waking hours. The most frequent time of day is the early morning, when the patient first awakes or while he is getting up. Many patients know that if they remain free from an attack until 8 or 9 A.M. there is no need to fear one during the rest of the day. Nocturnal epilepsy may remain unrecognized for years in the case of patients who sleep alone, the occasional wetting of the bed being diagnosed as nocturnal enuresis.

Epileptic attacks commonly cease for a number of years, quite apart from the effects of treatment, especially between infancy and the age of puberty; this renders the effects of treatment difficult to gauge.

Status epilepticus.—This term is applied to the condition in which a patient passes from one attack into another without recovering

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consciousness ; it may last for hours or days. It may be the first manifestation of the epileptic tendency, or be provoked by the sudden cessation of bromide treatment, or occur without obvious exciting cause. In severe and prolonged cases it is associated with a gradual rise of temperature and increasing rapidity of pulse and respiration. Its mortality is high.

Interparoxysmal state.—Epileptics show no physical signs by which their disease can be diagnosed. Most patients present some vaso-motor disturbances in the form of cold, purple extremities, and many suffer from chilblains. Not infrequently they are subject to migraine, and sometimes a liability to migrainous attacks has been replaced by a similar liability to epileptic seizures. The pupils of epileptic patients tend to oscillate on exposure to light, but neither this nor the vaso-motor phenomena can be regarded as characteristic of their disease.

There is an enormous variation in the mentality of epileptics. Although the disease is frequently associated with idiocy or mental deficiency, epileptic attacks may occur in persons whose mental equipment is up to, or even above, the average. We must distinguish, moreover, between the defective epileptic child and the child who starts with a normal intelligence and only becomes defective after repeated epileptic seizures. Mental deterioration is the inevitable result of frequently repeated attacks, whether they are of the major or the minor variety. At the same time it is difficult to apportion the blame for this deterioration between the attacks and their effect upon the outlook, the environment, and the education of the victim.

Attempts have been made to define an epileptic personality embodying self-complacency, egotism, exaggerated sensitiveness, suspicion, and inaccessibility, combined with a tendency to sudden outbursts of temper or suppressed irritability. But here there is difficulty in discriminating between causes and effects. Depression, irritability, and loss of memory and attention are frequently observed in the intervals between the attacks, and, as the latter become shorter, they remain as an integral part of the patient's character. In course of time he becomes less receptive and less expressive ; his thoughts and speech are characterized by a monotony of tone, and he sinks gradually into a condition of dementia. In this state he may be subject to accessions of violence or of immoral conduct.

Mortality.—Exact figures in regard to the

mortality of epilepsy are wanting, but there can be no doubt that only a small minority of patients die from the direct results of the disease. Status epilepticus accounts for most of these, asphyxia from turning over in bed or immersion in a bath during a fit for a few cases, and exhaustion due to maniacal outbursts for the remainder.

Diagnosis.—The diagnosis of epilepsy cannot be made from the physical examination of a patient between his attacks. Characteristic major attacks are not likely to pass unrecognized unless they are always nocturnal, when the occasional discovery of a blood-stained pillow, a wet bed, or a bitten tongue should arouse suspicion. Minor attacks escape notice more from ignorance or inattention on the part of a parent or a doctor than from any real difficulty in diagnosis. The repeated occurrence of similar phenomena, however transient, noticed or unnoticed by the patient, apparent to his companions, always sudden and generally without recognizable cause, should bring epilepsy at once to the mind of a medical man. They are often regarded as faints, but true *syncopal attacks* arise from some definite cause, such as a heated atmosphere, an emotional stimulus, or great fatigue, and are preceded by an appreciable period of faintness, generally associated with pallor and a feeling of nausea and "swimminess." They lack the precipitancy of epileptic phenomena. Attacks of *aural vertigo* are sometimes difficult to distinguish from epilepsy ; but they are associated with rotatory sensations, and their origin can generally be determined owing to the accompanying tinnitus and deafness. Major epileptic seizures may be simulated by *hysterical fits*, and it is always necessary to bear in mind the frequent occurrence of hysterical spasms as an immediate sequel of a transient and unrecognized petit mal. In hysterical fits the patient is not convulsed but struggles, the lips may be bitten but not the tongue, and the sphincters are not relaxed. The patient is flushed rather than livid, and the attack tends to be of much longer duration than that of an epileptic convulsion.

The diagnosis from organic brain lesions, such as intracranial tumour, cerebral syphilis, chronic lead-poisoning, arterio-sclerosis, or uræmia, can be made only by careful physical examination between the attacks, and by attention to the history of the onset and course of the disease.

Prognosis.—Only general and somewhat

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vague statements can be made in regard to the prognosis in epilepsy. Early recognition and treatment are certainly the most favourable omen in subjects who are otherwise healthy. An hereditary taint is not altogether an unfavourable factor in prognosis. Cases which relapse after successful treatment has been discontinued rarely do well even when the treatment is renewed under identical conditions. The age of onset is not of great importance, but the later fits appear up to 30 years of age the better the outlook. Petit mal defies treatment more often than grand mal, and the occurrence of attacks both by day and by night is less favourable than when they are entirely nocturnal or entirely diurnal. Patients with much mental deterioration do not respond well to treatment, but spontaneous remissions may occur.

Treatment.—When a tendency to epilepsy has been determined, the first principle should be to interfere as little as possible with the ordinary routine of the child's life. It is neither necessary nor wise to stop all work, but those responsible for his education should be instructed that exposure to strain must be avoided. It is equally important to prevent the child from feeling that he is different from other children, that he is more or less under suspicion, and that he is constantly being restrained from sharing in the pursuits of his brothers and sisters. Any tendency to introspection should be countered, and fears allayed by frankness and encouragement. It is better to take certain inevitable risks than to let him brood over his disability.

General hygiene.—All epileptic patients should have plenty of fresh air and should avoid the stuffy atmosphere associated with picture palaces, theatres, and many churches. They should be protected from excitement without undue interference with their pleasures. Outdoor games are better than indoor parties. Long hours of sleep must be encouraged, and all sources of great fatigue avoided. Adults must be forbidden such occupations as driving a car, as the occurrence of an attack might endanger the lives of others. The risks of bicycling or of riding may often be taken with advantage. Regular employment of a congenial nature is of the utmost importance, the greatest curse of the epileptic being his inability to procure it or to keep it. Minor disturbances of health must be attended to, especially those associated with errors of refraction, nasal defects, tonsils, adenoids, or the

functions of the gastro-intestinal tract. Diet should be regulated in accordance with the patient's idiosyncrasies, and heavy meals, especially in the latter part of the day, avoided. It is not necessary to stop all meat or all salt, but moderation in regard to both should be enjoined. Alcohol must be left severely alone, and tobacco enjoyed only in small quantities.

Medicinal.—No experienced observer can doubt that the course of epilepsy is largely influenced by habit. In other words, every seizure, however slight, helps to establish more firmly the "fit habit" of the brain. The use of drugs in the treatment of epilepsy is mainly directed towards the prevention or cure of this habit.

The value of bromides has stood the test of sixty years' experience and, in spite of popular and even professional prejudice and ignorance, is still established in an impregnable position compared with that of any other therapeutic agent. When they fail in completely controlling the occurrence of fits, the patient's relatives are apt to ascribe to the drug the mental deterioration which is the inevitable result of the recurring attacks; but every physician with large experience knows that patients who take moderate doses of bromide daily with successful results can continue the drug year after year without the slightest sign of mental deterioration. Frequently, indeed, patients become brighter, less depressed, more energetic, and better in general health after the administration of bromides. Professional prejudice against their use has arisen largely from ignorance of the way in which they should be employed. The essential for success is regularity and continuity in administration. It does not matter which of the bromide salts is used, but a medical man should choose one and keep to it. There is no advantage in prescribing a mixture of three salts, and there is no valid reason for supposing that one salt is more depressing than another. If a drachm of bromide daily is unsuccessful in controlling epilepsy it is unwise to try bigger doses; in the large majority of cases 30 or 40 gr. per diem will produce the maximal effect, and in many cases 15 or 20 gr. are sufficient. Never give two doses daily when one is sufficient, and never give three doses daily if this can be avoided. Continuous treatment is much more likely to be persevered in when the drug has to be taken only once or twice in the twenty-four hours. The medicine should be

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taken just before the time at which attacks are most likely to occur—in nocturnal epilepsy at bedtime, while if fits occur in the morning the dose should be taken immediately on waking. It may be wise to increase it during the week of menstruation if attacks are particularly prone to occur at that time. Two or three minims of liquor arsenicalis should be prescribed with each dose, and the efficacy of the mixture may be increased in many cases by the addition of tincture of belladonna, tincture of digitalis, tincture of nux vomica, borax, calcium lactate, or zinc salts. Only experience can enable one to judge how far one of these adjuvants is likely to be of use.

The administration of bromides cures a few cases, relieves a very large number, and is useless in a small minority. The amount of relief is variable, but when it enables the patient to carry on an employment or to live a comparatively normal life its importance is very great. If treatment partially controls the frequency of attacks it should be continued indefinitely. When attacks cease under the influence of bromides it is wise to continue their exhibition for several years, the dose being gradually reduced after two or three years of immunity from attacks. In such cases the medicine is doing no harm, and it is much wiser to delay its cessation than to risk a relapse.

Treatment of the epileptic attack.—In a few cases the patient can abort attacks when the warning is sufficiently prolonged, and each has his own particular method, learned by experience. He is not always successful, and no reliance can be placed on his efforts. During an attack the patient should be protected from injury, from biting his tongue, and from suffocation. No other measures are necessary, and it is advisable to allow him to sleep undisturbed if he passes from unconsciousness into a slumber. A post-epileptic hysterical fit can be cut short by any of the methods usually employed in dealing with such attacks.

In *status epilepticus* chloroform should be administered in order to stop the convulsions and to enable other measures to be taken against their renewal. An injection of hydrobromide of hyoscyne $\frac{1}{10}$ gr. should be given subcutaneously, and $\frac{1}{2}$ dr. of chloral hydrate placed in the rectum. The same channel should be used for the administration of nutriment, and a rise of temperature should be met

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by cold sponging or immersion in a cold bath in the case of children.

Marriage.—The majority of epileptics marry in spite of advice to the contrary, and marriage has no definite effect on the course of the disease. No support can be given to the idea that the condition of the patient is materially altered by either sexual intercourse or continence. Confirmed epileptics should not marry, on account of the hereditary nature of their instability; but this rule may be modified in the case of some patients who have acquired the epileptic habit after head injuries or brain disease. The recurrence of epileptic attacks is a grave and frequent source of marital unhappiness.

Institutional treatment.—Epileptic colonies are of great service in promoting the health of their inmates, because here they secure favourable surroundings, freedom from the psychological influences engendered by the struggle for existence under handicap, regular employment, and the social intercourse which is often denied them in the outer world. The fact that a patient lives under these conditions should not preclude him from careful individual attention and medicinal treatment.

E. FARQUHAR BUZZARD.

EPILEPSY, JACKSONIAN.—Jacksonian epilepsy is a term applied to convulsive attacks beginning in a particular region, and spreading along a definite line of march corresponding to the anatomical arrangement of centres in the cerebral cortex. In these attacks there is either no loss of consciousness, or unconsciousness only supervenes when the convulsions have become generalized. Jacksonian epilepsy is a symptom, not a disease. It is a result of a focal structural lesion in the brain which may be fixed and permanent, such as a scar resulting from inflammation or injury, or progressive, such as tumour or abscess. The diagnosis is that of the underlying conditions, and the treatment is that of idiopathic epilepsy supplemented by measures, medical or surgical, which aim at the removal of the disease. It must not be forgotten, however, that the excision of a tumour, for instance, may not bring about a cessation of attacks, and that medicinal treatment is necessary for prolonged periods after a successful surgical operation.

E. FARQUHAR BUZZARD.

EPILEPTIC IDIOCY (see EPILEPTIC INSANITY).

EPILEPTIC INSANITY

EPILEPTIC INSANITY.—The causes of the mental symptoms of epilepsy differ in no way from those of the somatic symptoms.

Symptoms.—Though the mental characteristics of epileptics, apart from episodal attacks, are often quite normal, and in rare instances surpass the average level of ability, yet very many patients exhibit some peculiarities. Epilepsy is particularly associated with all degrees of mental defect, from a level of complete ineducability to one at which it can only be said that the patient is rather backward. At any of these levels there will probably be found singular manifestations of vanity, selfishness, idleness, and religiosity, and, as an accentuation of these characteristics, a marked tendency to alcoholism, crime, and various sorts of mental disorder. In addition, however, to these features there are episodal mental symptoms associated with the fits, or occurring independently of them, having certain peculiarities which suggest that they are in the mental field analogous to the somatic symptoms, the so-called psychic equivalents. Lastly, there is the general mental enfeeblement which in many cases sooner or later makes its appearance, and which is known as epileptic dementia.

Considering first the symptoms associated with the fits, we find that some occur before the fit, some during it, and some after it. The approach of fits is occasionally announced by some change in the patient's character. He may for some varying period be depressed or excited, or rather stupid or obstinate, or be occupied by some particular line of thought. Occasionally an hallucination of some special sense may serve as an aura. Such hallucinations may be quite vague, or may be of definite sounds, sights, smells, and tastes. In some cases patients have asserted that when the fit has commenced they have not completely lost consciousness, but have been through a vague, dreamy state without marked affective tone, while others have experienced feelings of extreme terror and horror.

The mental symptoms succeeding a fit may consist in confusion, in which the perception of surroundings is most imperfect and a perverted judgment may lead to the commission of foolish or criminal acts. As an extension of this confusional state there may be a condition of automatism in which the patient may perform a series of acts, simple or complicated, of which he has no recollection when he "wakes up." In a few cases excite-

ment and violence are so extreme that the term "furor" has been applied to the state. Psychic equivalents are abnormal mental states occurring in epileptics, which come on with great rapidity, usually last but a few hours or days, and rapidly subside. Sometimes such attacks alternate with fits or occur in persons who have had fits, and in such cases there is but little doubt of their epileptic source. In other cases there have been no fits, great or small, and the epileptic character of the equivalents is deduced from the sudden onset, short duration, rapid disappearance, and other characteristics shortly to be described, and perhaps from a faulty family history. When the condition is established the patient is in a state of extreme confusion, and seems to be living in a world apart. He may give evidence of delusions which may be grandiose, persecutory, or hypochondriacal, and may exhibit conduct wholly inappropriate to his surroundings. It is of great importance to bear in mind that the actions of persons in this state are extremely impulsive and violent, and that they are in consequence among the most dangerous of all the mentally unsound, and may commit homicide, or sexual or other assaults of a determined nature. Associated with the attack, or occurring after it, have been noted such signs as tremors of the hands, face, and tongue, mumbling speech, staggering gait, and perhaps even inability to stand. Such signs, together with the impulsiveness and violence, suggest the epileptic origin of the attacks. After the attack is over there is forgetfulness, partial or complete, of that which occurred during its course.

Whether as a consequence of the fits or as a terminal feature of a general process, mental enfeeblement sooner or later appears. It is of very gradual onset, but its pace is liable to occasional acceleration, especially at such times as puberty and the menopause. The symptoms first show themselves as slowing of the mental operations and sluggishness of the reactions, combined with a lowering of the level of character, so that the patient becomes cunning and selfish. Sexual impulses are also liable to become overwhelming, and may lead to assaults, or perversion, or bestiality. As time goes on, mental deterioration increases, until at last the patient is left with a few elementary reactions.

Treatment of the mental symptoms is fundamentally the same as for other forms of epilepsy. The impulsive and violent character

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of the reactions must always be remembered, and an efficient staff of nurses or attendants obtained. The attacks are usually of short duration, and for this reason certification may not be necessary, always provided that the patient can be properly looked after elsewhere than in an asylum.

E. D. MACNAMARA.

EPIPHORA.—The term epiphora denotes an overflow of tears in sufficient quantity to trickle down the cheek instead of being carried away by the lachrymal channels into the nose. It is due to a variety of causes to be mentioned later, but the condition is always aggravated by exposure to wind and cold, and indeed seldom occurs indoors.

The ordinary moisture of the conjunctival sac and the eyeball is maintained by secretion from the Meibomian and other conjunctival glands, and receives nothing from the lachrymal gland, which is only called into action by the irritation due to the presence of foreign bodies or under the influence of emotion, dust, wind, or cold; therefore the lachrymal passages and duct are never used except under these conditions. The duct has only a potential cavity, and in addition to the soft parts has a definite bony boundary, which still further reduces its calibre, so that it is capable of little enlargement. The tears are carried away by means of a muscular mechanism in which the orbicularis and tensor tarsi muscles take the chief part.

Etiology.—The causes of epiphora are—

- (a) Excessive secretion due to irritation of the lids and conjunctiva.
- (b) Want of proper apposition of the punctum to the globe.
- (c) Obstructions in various parts of the lachrymal canal, e.g. small punctum, constrictions in canaliculus or lachrymal sac, or dust in any part of the canal.

Diagnosis.—The first point to determine in any case of epiphora is whether the duct is patent or not; this can easily be decided by using the two methods described under EYE. **EXAMINATION OF,** i.e. the use of a drop of fluorescein and syringing. On passing the nozzle of the syringe, one soon discovers any obstruction in the punctum and canaliculus, and the constriction of these soft parts can sometimes be overcome by dilatation with a conical Nettlehip probe. If the fluid ejected from the syringe passes freely down the nasal duct into the pharynx, nothing further is

gained in this direction, as we have demonstrated that the duct is sufficiently patent for the passage of tears.

If the fluid does not pass into the throat, the obstruction is usually in the duct, and probably at its nasal end. My belief is that nearly all cases of obstruction are due originally to disease of the nasal passages which blocks the nasal end of the duct, and that therefore the nose should be examined carefully before any further treatment is carried out. Epiphora may continue as a watery discharge, or the secretion retained in a dilated lachrymal sac may become infected by micro-organisms and be rendered muco-purulent. Again, a definite abscess may form and appear as an inflamed tender swelling at the inner canthus and over the neighbouring part of the nose, eventually pointing below the internal tarsal ligament.

Prognosis is not very satisfactory, for, unless the normal passage is re-established in some way, watering of the eyes in the wind and cold is certain to remain, and re-establishment is difficult to obtain in all but the milder cases and those due to congenital stenosis.

Treatment.—All treatment is directed towards keeping open the passage between the punctum at one end and the nasal opening at the other, and removing all muco-purulent discharge. Repeated syringing two or three times a week, with occasional injection of some astringent such as zinc or protargol, will often get rid of the discharge, and thus the inflammatory swelling of the soft parts caused by this constant source of irritation will subside and a passage be re-established.

Operations through the nose have been performed for enlarging the duct, e.g. West's operation.

A small punctum may be enlarged by removal of a small triangular piece of its conjunctival boundary, and excessive secretion due to inflammation of the conjunctiva may be treated with astringent lotions.

If these methods of treatment do not bring about a cure, the probability is that there is permanent thickening of the mucous membrane lining the nasal duct, and the passing of probes has then to be considered.

The old treatment of gradually enlarging the lachrymal duct by the passage of probes of varying thickness until one of very large size could be passed is quite out of date. It can only be accomplished by slitting up the canaliculus, rupturing the soft parts, and crushing the bone. Even if a passage is eventually

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established, at the expense of much severe pain and repeated probings, it can only be kept open by the periodical passing of a large probe or by wearing some form of cannula. Such a procedure is not tolerated permanently, and later the obstruction returns in an aggravated form due to callus and fibrous thickening.

The only indications for the passage of a probe, and then only the finest that is made, are in congenital stenosis and in some cases at an early stage when the obstruction is likely to be due to a plug of mucus.

Treatment is largely dependent on the amount of actual discomfort which the patient experiences; many cases require no treatment at all, since it is no great hardship to suffer slightly from watery eyes in the wind or cold.

If an abscess forms it must be treated like any other abscess, viz. by fomentations, and by an incision followed by packing the cavity with strips of gauze to encourage healing from the bottom. In troublesome chronic cases, and after the formation of an abscess, the best treatment is excision of the sac, as any pus-containing cavity near the eye is a danger in the event of an injury to or abrasion of the cornea; but for this operation an expert opinion is advisable. MALCOLM L. HEPBURN.

EPIPHYSITIS.—By epiphysitis is meant acute or chronic inflammation, not only of an epiphysis but of the bone in immediate relation to it.

Etiology.—The *acute* variety is usually the result of congenital syphilis, and is seen, as a rule, during the first three months of life; occasionally it is caused by pyogenic bacteria in cases of pyæmia. *Chronic* inflammation of an epiphysis is due to tuberculosis. Both varieties are not uncommonly preceded by a slight injury.

Pathology.—In acute cases the changes are those of osteo-chondritis affecting the diaphysis and the epiphysis where they are related to one another; the inflammation may extend for some distance into the diaphysis. The para-epiphyseal calcified cartilage becomes dull, opaque, and friable, and the cartilage may even be destroyed and replaced by granulation tissue, leading to separation of the epiphysis. Suppuration occurs and may extend to the adjacent joint as an acute arthritis.

Symptomatology.—The epiphyses most commonly affected are those related to the knee-joint, the elbow, and the wrist; in *acute* epiphysitis several bones are often involved at the same time, and a symmetrical distribution

is common. The end of the bone becomes enlarged, painful and tender, and the infant does not move the affected limb, giving the typical picture of syphilitic "pseudo-paralysis"; this is especially seen in those cases where spontaneous separation of the epiphysis has occurred; palpation or movement causes the infant to scream, and deformity or abnormal mobility may be detected. If the joint is involved it becomes acutely inflamed, hot, swollen, and tender, and is held in a semiflexed position. The distension may lead to pathological dislocation; when this occurs in the knee, the tibia is flexed, externally rotated, and displaced backwards and outwards. As a late result of the disease, diminution or cessation of growth of the bone may occur, and deformity will result if the epiphysis is detached. Arthritis may lead to ankylosis, which is usually of the fibrous variety.

In pyæmia early involvement of the adjacent joint occurs, and the main symptoms are those of suppurative arthritis. The causative bacteria may be staphylococci, streptococci, or pneumococci.

In the *chronic* variety the disease usually affects older children; under treatment the inflammation may become quiescent and eventually cured, but usually caseation ensues and the neighbouring joint is involved; this process is considered under ARTHRITIS, TUBERCULOUS.

Diagnosis.—Syphilitic epiphysitis must be diagnosed from *rickets*, in which the disorder is confined to the ends of the bones, with no extension to the diaphyses; rickets usually occurs at a later age than acute epiphysitis. *Traumatic inflammation* of a joint or the end of a bone may simulate epiphysitis. *Scurvy*, too, is sometimes simulated, but this again is seldom met with until after the age of six months, and it may be distinguished by the presence of other scorbutic signs, such as hæmorrhagic gums, petechiæ, and hæmaturia, and by a history of faulty feeding.

The multiplicity of the lesions, their symmetrical distribution, the family history, and signs of syphilis elsewhere in the body will make a diagnosis possible in most cases; in early or doubtful ones a Wassermann test may be done. X-ray examination shows irregularity of the epiphyseal line and alterations in the bone structure, especially in the end of the diaphysis; it is chiefly of value in excluding an incomplete fracture.

Prognosis.—Syphilitic epiphysitis soon sub-

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sides under antisyphilitic treatment, but in nearly all cases some deformity will result from interference with the growth of the bone. The prognosis of the other forms is considered under ARTHRITIS, TUBERCULOUS, and ARTHRITIS, SUPPURATIVE.

Treatment.—In the syphilitic variety the limb should be immobilized by a light plaster-bandage splint; if the epiphysis is detached it should be reduced under an anæsthetic before the splint is applied. If a joint is greatly distended it should be emptied by aspiration. Mercury must be given, either by the mouth in the form of hydrargyrum cum creta $\frac{1}{4}$ -1 gr. t.d.s., or by inunction, a piece of blue ointment the size of a pea being applied to the binder or rubbed into the skin each night. The mother must be treated actively by salvarsan or one of its substitutes, and by mercury. The treatment of the acute non-syphilitic forms is that of suppurative arthritis and osteo-myelitis, and of the chronic forms that of tuberculous arthritis (*see* ARTHRITIS, SUPPURATIVE; OSTEO-MYELITIS; ARTHRITIS, TUBERCULOUS; PSEUDOCOXALGIA).

C. W. GORDON BRYAN.

EPISCLERITIS (*see* SCLERA, AFFECTIONS OF).

EPISPADIAS (*see* HYPOSPADIAS AND EPI-SPADIAS).

EPISTAXIS (Nose-bleeding).—The causes of epistaxis may conveniently be divided into local and general, though it should not be forgotten that a general cause may coexist with a local lesion. Its frequency is explained by the abundance of fragile and delicate blood-vessels in the nose.

Local causes.—The hæmorrhage produced by these is usually small in amount, though occasionally considerable. It may be due to the congestion of the mucous membrane which accompanies hypertrophic rhinitis, nasal spurs or adenoids, or may follow ulceration produced by diphtheria, syphilis, new growth, or foreign bodies. A small ulcer situated near the front of the septum is a common cause of repeated bleedings, and is often overlooked.

Unilateral epistaxis indicates a local cause, and should always suggest the probability of ulceration. When the bleeding is from a focus far back in the nose or occurs during sleep, hæmoptysis or hæmatemesis may be simulated. Trauma, by a blow on the nose or by causing fracture of the cranial base, is another cause.

General causes.—There are many people who are subject all their lives, though more so in childhood, to epistaxis, the explanation of which cannot be found in any local lesion or constitutional state. Plethoric persons, too, are liable to bleeding from the nose on slight exertion, during excitement, or after taking alcoholic liquors. Of more importance is the bleeding which results from high arterial tension in arterio-sclerosis and especially in Bright's disease, for it is a warning of the advent of cerebral hæmorrhage and of the need for preventive measures. On this account the urine of patients past middle life who suffer from epistaxis should be examined. Hepatic cirrhosis is another frequent cause. Epistaxis sometimes occurs in the early stages of specific infections, especially in typhoid fever, rheumatism, and influenza, and also during the course of the hæmorrhagic forms of the exanthemata. It is a common accompaniment of blood disorders: hæmophilia, anæmia (especially its pernicious form), leukæmia, lymphadenoma, and splenic anæmia of both the infantile and adult forms, purpura, scurvy, and toxicæmic jaundice. Among mechanical causes may be mentioned the local congestion due to mitral disease, and the turgescence produced by the paroxysms of whooping-cough or by suffocation. Rarefaction of the air, as encountered in mountain-climbing or in aviation, is sufficient to cause epistaxis in some subjects. In neurotic women it is occasionally regarded as vicarious menstruation.

Treatment.—For slight epistaxis no treatment is necessary to control the bleeding, and treatment with this end in view is contra-indicated in plethora, arterio-sclerosis, and Bright's disease unless the patient shows symptoms attributable to loss of blood. But a careful examination should always be made to discover the cause, and the nasal cavities scrutinized if the cause is not obvious. For an attack of bleeding the patient should be kept recumbent and motionless, with the head and shoulders high. The arms may be raised above the head, and ice applied to the nares or in the region of the lower cervical spine may be effectual. Very cold or very hot nasal douches are sometimes successful. Astringents such as perchloride of iron, alum, acetate of lead, hamamelis may be used, but generally fail; perhaps the most successful local application is adrenalin (1-1,000), or a saturated solution of calcium chloride. If a bleeding-point is discovered it may be touched with

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chromic acid or silver nitrate, or, better, with an electric cautery at a dull-red heat or a probe heated at a spirit lamp, a 10-per-cent. solution of cocaine having previously been applied after all bloodclot has been removed by douching. In obstinate cases the posterior nares should be plugged, preferably with gauze lubricated with oil to avoid recurrence when the plug is removed. The plugging should be done through the anterior nares, a strip of gauze about 18 in. long and $\frac{1}{2}$ in. wide being packed firmly in against the bleeding-point. The introduction of special nasal distensible bags is an alternative and improved method. The plugging or bag should be removed in about twenty-four hours. When epistaxis is recurrent, perchloride of iron or calcium chloride given by the mouth often reduces the number and severity of the attacks. In the blood diseases the injection of normal horse-serum or transfusion may be indicated for intractable epistaxis; they are of considerable value.

FREDERICK LANGMEAD.

EPITHELIOMA (see SKIN, MALIGNANT GROWTHS OF)

EPULIS.—A tumour growing from the periosteum of the jaws. It may be either a fibroma (fibrous epulis), a slow-growing sarcoma often myeloid in type (malignant epulis), or, sometimes, a true myeloma. The tumour varies in size. Usually it presents itself as a small flattened pale growth attached to the gum between two adjacent teeth, which are separated by it. It may spring from a tooth-socket. It is firm to the touch. On account of their malignant tendencies wide removal is necessary. Both the involved teeth should be extracted and a V-shaped portion of jaw sawn out with the tumour attached.

C. A. PANNETT.

ERB'S PALSY (see SPINAL NERVES, LESIONS OF).

ERB'S SYPHILITIC SPINAL PARAPLEGIA (see CEREBRO-SPINAL SYPHILIS).

ERGOTISM (see POISONS AND POISONING).

ERYSIPELAS (ἐρυθρός, red; κέλλα, skin).—A contagious disease characterized by a spreading inflammation of the skin or mucous membrane associated with general febrile disturbance, the result of infection with a specific micro-organism. The term erysipelas should be reserved for inflammation of the cutaneous

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and mucous integuments, inflammation of the underlying areolar tissue being known as cellulitis.

Etiology.—Experimentally, a spreading inflammation of the skin has been produced by the inoculation of such organisms as streptococci, pneumococci, *B. coli* and even staphylococci, but in man cases of erysipelas are almost invariably the result of a streptococcal infection. Hence erysipelas may briefly be defined as streptococcal dermatitis. Much of our knowledge of erysipelas is due to Fehleisen, who in 1882 isolated from a case of this disease a streptococcus to which he gave the name of *Str. erysipelatis*. By inoculation experiments with this organism he was able to reproduce the disease in man and in lower animals. There has been much discussion in the past as to whether *Str. erysipelatis* is bacteriologically identical with *Str. pyogenes*. It is a noticeable fact that erysipelas passes from patient to patient as erysipelas, and that purulent conditions due to *Str. pyogenes* are not likely to be followed by erysipelas. A well-established clinical connexion exists between erysipelas and puerperal septicæmia. The truth appears to be that the *Str. pyogenes* of the older bacteriologists was not an entity. It is now known that the organisms responsible for erysipelas belong to the hæmolytic group of streptococci, and, moreover, many different strains occur, a circumstance which explains the want of conspicuous success in the treatment of this disease by stock serums and vaccines. Similar, if not identical, hæmolytic streptococci are concerned in many cases of puerperal septicæmia.

Fehleisen showed that the constitutional effects in erysipelas are to be attributed to the absorption of toxin rather than to a blood infection. Having induced experimental erysipelas by injecting streptococci into the ear of a rabbit, he proceeded to amputate the ear below the inflamed area by means of a Paquelin's cautery, with the result that the temperature fell and the constitutional symptoms abated. It would appear that it is only when erysipelas occurs as a terminal infection, and in fatal cases, that a true septicæmia arises in this disease.

Among predisposing causes must be mentioned unhygienic conditions such as obtained in pre-antiseptic days in many hospitals, where the accumulation of dirty dressings and the ignorance on the part of doctors and nurses of the source and paths of infection were largely responsible. Nowadays the chief predisposing

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conditions are to be sought in the medical history of the patient. Thus erysipelas is not uncommon in persons recovering from a debilitating disease like typhoid or suffering from chronic ill-health as in chronic Bright's disease, diabetes, or alcoholism.

The immediate exciting cause is the entrance of streptococci through a wound. Holmes has pointed out that just as pneumococci can be found in the mouths of many healthy persons, so streptococci may be present in the nose, nasal fossæ, and middle ear without giving rise to active trouble. He regards the so-called idiopathic facial erysipelas as usually originating in, and extending from, the nasal cavity, a slight abrasion within the nostril being the common site of entry for the organism. In some cases the infection apparently spreads through the whole thickness of the tissues of the nose to reach the skin. Subjects of chronic coryza with abrasions about the nostrils and lips would appear to be particularly prone to the disease. The inflammation does not always start exactly at the edge of the abrasion, and the same phenomenon has been observed in experimental cases. The term idiopathic has been applied to cases in which no definite portal of entry can be found, but there is no essential difference between such cases and those in which a definite wound exists. The breach of surface may be microscopic and impossible to locate, and it is quite possible for a slight scratch to heal during the incubation period of the disease and in this way to escape detection. In addition to the nose and lips, other favourite positions for the disease to attack are the external auditory meatus, the inner angle of the eye, and the edge of the scalp. Wounds such as those caused by the introduction of Southey's tubes into œdematous legs are especially likely to become the starting-point of infection. Burns, scalds, or the lesions of vaccinia and variola may constitute the portal of entry in other cases.

Pathology.—Once the organism has obtained an entry, rapid extension by way of the lymph spaces and lymphatics ensues. In this way an œdematous lymphangitis accompanies the inflammatory hyperæmia. A section through the skin shows a dense cellular infiltration in which lymphocytes frequently preponderate, while the lymphatic spaces at and beyond the spreading edge are seen to be crowded with organisms. The reason the inflammation remains limited so frequently to the skin appears to be that the dermal lymph-

spaces constitute for it the path of least resistance, so that the micro-organisms find it more difficult to travel in any other direction. Cellulitis results if the organisms spread into the subcutaneous tissue, while septicæmia or pyæmia will follow their successful invasion of the blood-stream. In fatal cases infarcts have been found in the lung, spleen, and kidney. French observers have laid stress on the abundance of the thin fluid exudate in erysipelas (which they often describe as an œdematous dermatitis) as contrasted with the richer fibrinous exudate met with in other forms of inflammation of the skin. The blebs which are not infrequently seen surmounting the affected area are further evidence of the associated œdematous lymphangitis. Where mucous membranes are affected a fibrinous layer usually covers the surface, while a mucopurulent discharge is not infrequent in addition. A polymorphonuclear leucocytosis occurs in the disease, its maximum coinciding with the acme of the fever.

Symptomatology.—After infection there is an incubation period of one to three days. Prodromata are rare. The mode of onset varies. Sometimes local manifestations may precede the constitutional disturbance, but frequently severe rigors and vomiting accompany the appearance of the eruption. The temperature quickly rises to 103° or 104° F., and the usual concomitants of fever, headache, anorexia, etc., appear. The pulse becomes full and bounding. Apart from wounds, the face is the region most frequently attacked. The affected skin becomes red, swollen, painful and tender. As the inflammation extends it is noteworthy that the spreading margin is well defined and projects above the level of the healthy skin. When taken between the fingers the affected skin can no longer be raised in folds. As the inflammatory œdema increases, the patch becomes tense and shiny, and in severe cases blebs containing turbid fluid make their appearance. When these burst the contents dry, to form yellowish crusts. The eyes may be completely closed by swelling of the eyelids, wrinkles become obliterated, the lips and ears thickened, and by the third day the features may no longer be recognizable. As the inflammation advances, the redness dies away from the part first affected. The inflammation fails in many cases to spread beyond those sites where skin and underlying structures are tightly bound together; thus it seldom passes beyond the chin. Swelling and

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tenderness of the lymphatic glands draining the affected area occur, and occasionally the glands suppurate. The inflammation commonly lasts for a week, and as it subsides the skin becomes flaccid, wrinkles reappear, and desquamation takes place. In cases in which a surgical wound is infected the wound becomes hot and dry, healing ceases, and a reddish blush spreads from the edges to the surrounding skin.

The temperature chart usually shows high fever at night, with morning remissions. In favourable cases the fever comes to an end about the sixth or seventh day. Fall by crisis is frequent. In the more severe cases, as the disease progresses the patient becomes increasingly restless, there is delirium at night, the tongue becomes dry and brown, and death is sometimes preceded by vomiting and diarrhoea. In erysipelas relapses are frequent, the tendency to recurrence being perhaps more common than in any other infective disease.

In addition to the acute form of the disease a more chronic variety occurs in which the inflammation tends to wander from one part of the body to another, the so-called *Erysipelas Migrans*. In this form fever is slight and constitutional symptoms are usually mild.

In newly born children the umbilical wound is the chief avenue of infection—*Erysipelas Neonatorum*. As Ballantyne has pointed out, the first signs of redness may be seen not round the umbilicus but near the symphysis pubis, as the infection often travels along the track of the hypogastric arteries. *Erysipelas neonatorum* is almost invariably fatal.

Various **complications** may be met with. The inflammation may spread to the larynx, producing oedema with its attendant dyspnoea. Should the trachea become involved, death from broncho-pneumonia is likely to result. If the eye is affected there result conjunctivitis with chemosis, keratitis, and possibly sloughing of the cornea. Suppuration of the middle ear may follow extension to that cavity. Diffuse cellulitis is not infrequent. In infants peritonitis has followed erysipelas of the anterior abdominal wall. Permanent oedema due to blocking of the lymphatic trunks occasionally ensues. Loss of hair may follow erysipelas of the scalp, but this is rarely permanent. Permanent loss of eyebrows is more frequent. While albuminuria is the rule in erysipelas, nephritis is fortunately a rare complication. In the latter case, examination of the urine shows the presence of casts and streptococci.

Death may occur from acute uræmia, or a chronic nephritis may be initiated. Among other complications occasionally arising must be mentioned meningitis, pleurisy, pericarditis, infective endocarditis, suppurative arthritis, and suppurative parotitis. The beneficial influence which erysipelas sometimes exerts upon cases of lupus and upon certain varieties of sarcoma is the foundation of Coley's method of treating inoperable cases of the latter disease.

Diagnosis.—Difficulty is not often experienced. The tense red patch—including no islands of healthy skin—painful and thickened right up to its well-defined margin, which projects above the level of the unaffected skin, and the accompanying fever and constitutional symptoms rarely leave room for doubt. When the scalp is attacked the colour is less vivid, but there is considerable oedema, and pain is more acute. In pharyngeal cases, in addition to the severe sore throat, there can sometimes be detected a purplish tint in the dark-red brawny discoloration of the mucous membrane. *Erythema*, especially perhaps when arising in the course of Bright's disease, is sometimes mistaken for erysipelas, but severe constitutional disturbance is lacking. In erysipelas the redness of the skin does not completely disappear on pressure as in erythema, and the patch, when taken between the fingers, cannot be raised in folds, but is resistant owing to the oedematous infiltration of the skin. An *acute eczema* occasionally gives rise to difficulty. But the redness, instead of showing a steady line of spread, almost at once involves a large area, and in place of the well-defined raised margin of erysipelas the redness blends almost insensibly with healthy parts. Fever is absent or very slight, and although vesicles may be present, in case of doubt a smear preparation or culture will settle the diagnosis, as streptococci are easily obtained from the erysipelatous area. The early stage of *herpes* occasionally suggests erysipelas, but the rapid appearance of vesicles and their characteristic distribution soon dispel doubt.

Prognosis.—Except at the extremes of life, erysipelas is not a very dangerous disease, and in previously healthy patients the mortality is very low. Most of the deaths ascribed to it have occurred in cases of chronic Bright's disease, diabetes, and chronic alcoholism, where it has arisen as a terminal infection. The outlook is serious when erysipelas follows upon the tapping of dropsical legs. Untoward

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incidents are high fever, delirium, severe vomiting or diarrhoea, and also the typhoid state.

Treatment.—The patient should be isolated, and if the nurse has any abrasion on her own hands, this must be suitably protected. A brisk purge should be given at the outset. The diet should be light and nutritious, and, as in other infective diseases, alcohol should only be given when some distinct indication for its use arises. The internal administration of drugs does not appear to cut short the disease, but *tr. ferri perchloridi* in $\frac{1}{2}$ -1-dr. doses every four hours is thought by some physicians to produce such an effect. Locally, the application of ichthyol and glycerin in equal parts is a useful measure. It is comforting to the tense, burning skin, and ichthyol appears to be especially inimical to streptococci. When there is great pain, lead-and-opium lotion will often relieve. Should diffuse cellulitis ensue, incisions may be necessary, and the assiduous use of an arm bath will repay the trouble involved. The application of brilliant-green has been advocated, and it is claimed that a dramatic disappearance of both rash and fever has occurred as early as the second and third day of the disease. A 5-per-cent. aqueous solution of brilliant-green is applied once daily in mild cases, twice a day in severe ones, and the painting should extend for an inch or so beyond the edge of the patch. The part should be kept covered with a piece of linen to prevent staining of the bedclothes, and a face mask used if the face is affected. The discoloration can afterwards be removed from the skin by bathing the part with a little weak alcohol and washing with ether soap. The older methods of applying silver nitrate around the area and of making incisions outside the affected zone to prevent spread have been largely abandoned owing to lack of success. An autogenous vaccine is worthy of trial in chronic cases of erysipelas migrans, or when frequent relapses have occurred. Antistreptococcic serum has, on the whole, proved disappointing.

C. E. LAKIN.

ERYSIPELAS MIGRANS (see **ERYSIPELAS**).

ERYSIPELAS NEONATORUM (see **ERYSIPELAS**).

ERYTHEMA.—This term, widely employed to indicate reddening of the skin, may signify a simple transient redness, such as may be produced by friction of the surface, or the

severest reactions produced by bacterial infection, such as erysipelas; it is also applied to certain special localized reddening of the surface usually associated with exudation, of which the various forms of erythema multiforme are examples. For convenience, however, it is advisable to consider the condition as being represented by two types—(1) the simple erythemas due to mechanical or physical causes producing redness, including the ordinary reaction of inflammation, and (2) certain special forms of localized character which may have a specific cause. Intermediate between these two more or less well-defined groups are particular forms of erythema, such as those distinguishing the specific fevers, and reactions that are produced by the introduction of foreign material such as antitoxic serum into the circulation. Erythemas of this kind have relations especially with the second group.

SIMPLE ERYTHEMAS

The redness of the skin is produced by dilatation of the capillaries owing to the special cause in operation. These causes may be of central origin acting under the influence of the stimulus produced by emotion; in other cases the reddening seems to be due to more local effects upon the vaso-motor mechanism such as may be produced by friction or injury. In both these cases the erythema is usually temporary and localized. Examples of generalized erythema are, however, not uncommon, and occur, for instance, as a result of gastrointestinal stimulation; thus there may be redness of the surface after a satisfactory meal or on taking food containing aromatic or pungent materials. In such cases the treatment of the erythema must depend upon the recognition and removal, if necessary, of the stimulating cause. Chemical irritants or foci of bacterial infection must be dealt with. In the majority of instances the vascular reaction is of transient nature, and of a degree so slight that no medical interference is called for.

Treatment.—The principle of treatment underlying all such conditions is to avoid all medicaments likely to macerate or soften the protective epithelial surface. Lotions, fomentations, or ointments are rarely necessary, and should be used with discretion. Cold to the surface is frequently comforting; it is for this reason that the application of ice and the use of evaporating lotions containing alcohol are sometimes of service. The application of



PLATE 11.—ERYTHEMA IRIS.

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dusting powders to the erythematous surface is often agreeable to the patient and useful in other ways. The powders should be of the finest quality and without grit, and should be so applied as to produce the thinnest possible layer; used in this way they may be employed repeatedly. A useful formula is—Boric acid 15 parts, zinc oxide 15 parts, siliceous earth (e.g. talc, "Cimolite," etc.) 70 parts. The boric acid is useful as an antiseptic, and the addition of small quantities of camphor or menthol may be beneficial on account of their antipruritic qualities.

The *intermediate group of erythemas* of the specific fevers, serum rashes, rashes following the use of enemata, etc., are usually distinguished from the foregoing group by the fact that they frequently show a certain amount of serous exudation in the reddened areas of skin. The serous exudation may be of sufficient amount to raise the horny layer of the epidermis and produce vesication. In some cases not only does the serum of the blood exude, but also red and white cells. The serum may be bloodstained, or the eruption may assume hæmorrhagic or purpuric characters. It will be recognized that this distinction is one of degree rather than of kind; the exudative features resemble those of the special erythemas.

SPECIAL ERYTHEMAS

The term erythema is frequently used in a special sense to indicate a group of eruptions with many varieties in which, in addition to reddening of the skin, frequently well localized, exudation of serum and of the blood-contents, and reaction of the tissues producing infiltration by new cells—leucocytes, connective-tissue cells, and cells of more specialized types—may occur. Perhaps the most common and the most easily recognized of these special erythemas is **erythema multiforme**, sometimes described as *erythema exudativum multiforme*. Though it may occur at all ages, it is more common in young persons. Frequently after some disturbance in health, localized patches of redness appear on the skin, often sparsely distributed, but sometimes in numerous areas. The reddening, commencing as a small point, spreads at the margin so as to produce rounded figures, and by the coalescence of neighbouring lesions the patches of erythema assume characteristic circinate outlines. The affected area is not only reddened, but raised owing to

serous exudation into the upper layers of the cutis and the epithelium. Frequently the horny layer of the epidermis becomes detached from the underlying germinating epithelium, so that vesicles, or even bullæ of considerable size, are formed. In some cases the central portion of such a lesion tends to subside, while the erythema spreads at the margin with swelling or vesication. In this way one or more circles of erythema may surround the original focus, which has gradually become pale while the successive margins have reddened. The margins themselves are usually well defined; outlying points of the erythema may, however, occur, giving rise to the formation of papules. The eruption may be distributed over any part of the surface of the body, and the mucous membranes occasionally; but very frequently it affects the extremities, often choosing the dorsal surfaces of the forearms, wrists, and fingers. Various names have been given to stages of this condition, and thus we have such well-known forms as *erythema vesiculosum*, *erythema bullosum*, and *erythema iris* (PLATE 11), this last name being given to the type of eruption with progressive circular margins.

The causes of this condition seem to be very various; many of the "drug" eruptions come into this category. It is suspected that in many cases the cause is a toxæmia of gastrointestinal or other origin.

Erythema nodosum is a special form of this condition, producing characteristic reddened and swollen bumps, especially on the extensor surfaces of the extremities. The affected areas vary much in size, from minute points to considerable patches several inches in diameter. They frequently produce much pain and tenderness during their formation, and even during their subsidence. This condition is frequently associated with pyrexial reactions, the temperature ranging up to 105° F. It was long thought that erythema nodosum had special relation to the infection of acute rheumatism; it is true that pain in, and even a certain amount of synovitis of, the joints may coincide with the eruption, but this and other types of erythema multiforme occur in cases where there is no definite evidence of rheumatic infection. The relationship to rheumatism is doubtful.

Erythema induratum (scrofulosorum); *syn.* Bazin's Disease.—This condition is perhaps the most specialized of the special forms of erythema. It occurs at all ages, but as a rule in

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younger people. The extremities are most frequently affected. It has a peculiar distinguishing feature marking it off from similar exudative erythematous conditions, viz. that parts of the affected areas tend to necrose, producing destruction of the epidermis, cutis, and subcutaneous tissues, and that from these areas of necrosis ulceration may spread. The anatomical lesions show not only serous or hemorrhagic exudation, but well-marked infiltration of the skin with cells, mainly of connective-tissue origin, of many different types. This cellular infiltration frequently includes, for example, well-formed giant cells in addition to other varieties. In cases of this disease there seems to be a gradual transition from those showing simple vascular reaction to those in which there is a well-organized cellular infiltration of the type indicated. It will be noticed from the description given that such cases deserve the name of *gumma* rather than of simple erythema; the lesions have often been mistaken for syphilitic gummata, and errors in treatment have consequently been made. A relationship of the disease to tuberculosis is, however, more possible; some cases described have no doubt been definitely subcutaneous or cutaneous tuberculosis. In a few examples tubercle bacilli are stated to have been discovered, and positive inoculation experiments on animals made. In still other cases with well-marked features of the disease, showing histologically the occurrence of the very special type of giant cell, no evidence of tuberculosis has been obtained by either clinical, serological, histological, or experimental methods. The exact etiology of this remarkable condition is still undefined.

Treatment.—From what has been said as to the different types and causes of erythema, it will be evident that no definite line of treatment can be laid down for all cases. Most important is it to find out as closely as possible what is the cause of the disease. Gastro-intestinal conditions must be investigated; in other cases sources of bacterial or other infection must be discovered, removed, or cured; in some cases specific diseases (for which we have special remedies) must be brought under control. Of drugs administered internally, apart from remedies administered for specific reasons—such as quinine, salicylic acid and salicin, mercury, and arsenic—arsenic and quinine have been most frequently used for their general effect. It must be admitted that these

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two drugs exhibited in this way have not often been found very successful. A more reasonable and successful method of treatment is to take all precautions to build up or restore the patient's general health. Locally, the most satisfactory methods are those of a protective and soothing nature. Soothing lotions of calamine and lead, soothing salves such as cold cream, zinc ointment, boric ointment, and the weaker salicylic acid ointments and creams, have great value. The drawbacks, which are considerable, are the difficulties in their proper application and the risk of macerating and producing further injury to already damaged superficial structures. When the lesions of the special erythemas have ulcerated they must be dressed on ordinary antiseptic principles; rest and protection are essential, as not infrequently ulceration is difficult to heal. Dusting powders of the type already mentioned will be found to be of very general utility; they should not be used on a broken or oozing surface without special precaution in the way of dressing by means of appropriate ointments, as they tend to produce crusts which retain infected discharge, and thus intensify the infective elements.

Occasionally the lesions of erythema multiforme affect the mucous membranes, especially of the mouth and throat; the vesicles and ulcers so produced are usually superficial but often very troublesome. In such cases careful treatment by means of appropriate mild antiseptic mouth-washes or the application of mild astringents is required.

JAMES GALLOWAY.

ERYTHEMA INDURATUM (*see* ERYTHEMA).

ERYTHEMA MIGRANS (*see* STOMATITIS AND GLOSSITIS).

ERYTHEMA MULTIFORME (*see* ERYTHEMA).

ERYTHEMA NODOSUM (*see* ERYTHEMA).

ERYTHEMA PARATRIMMA (*see* BED-SORES).

ERYTHEMA PERNIO (*see* CHILBLAIN).

ERYTHRÆMIA (*see* CYANOSIS).

ERYTHROMELALGIA. — Red neuralgia of the extremities—a condition first described by Weir Mitchell. Pain, usually in the foot and toes, sometimes in the hands, is rapidly

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followed by a flushing of the extremities and swelling, with a subjective sensation of considerable heat and discomfort. This condition is likely to appear in the foot after walking, especially after fatigue, and the symptoms may become much aggravated when the extremities are allowed to hang down. The arteries pulsate visibly, and the veins of the dorsum of hand and foot become much swollen, while the extremity changes colour from a pink to a slightly purplish hue, frequently with small blotches of deeper discoloration. Cyanosis does not become so pronounced as in Raynaud's disease, nor is there the tendency to local gangrene and loss of tissue which is seen in that affection.

The disease is a rare one, to which both sexes are liable; nervous instability and neurotic heredity are certainly predisposing factors. Many cases have been described as complicating certain chronic spinal diseases, such as disseminated sclerosis and tabes dorsalis. When it is independent of such spinal diseases there is no anaesthesia nor trophic lesions of the skin, and the reflexes are normal. Prickling sensations on the feet or hands when the limb is swollen, red, and painful are not uncommon, and the pain may be agonizing in its severity.

Diagnosis.—There are several conditions from which erythromelalgia must be differentiated. *Raynaud's disease* may be distinguished by the deeper and more chronic cyanosis, with coldness of the extremities and tendency to peripheral gangrene. *Acroparæsthesia* is common in the hands and does not show the distinctive redness and swelling, nor the same severity of pain. *Chronic obliterative endarteritis* causes almost persistent pain without paroxysmal turgescence, and the cyanosis is patchy, deeper in colour, and frequently leads to extensive gangrene. *Chilblains* and other vaso-motor angio-neuroses may also have to be distinguished from erythromelalgia.

Treatment.—In severe attacks, rest is necessary, and the limb should be kept raised, while for severe pain the coal-tar analgesics and hypnotics must be given, such as 7 gr. of antipyrin or pyramidon with 2½ gr. of medinal, repeated three or four times a day. Local arm or leg electric baths may be used with advantage in less severe attacks, the constant galvanic current being employed.

WILFRED HARRIS.

EXOPHTHALMIC GOITRE

ESPUNDIA (see ORIENTAL SORE).

ESTHIOMÈNE (see VULVA, DISEASES OF).

ETHMOIDAL CELLS (see SINUSES, ACCESSORY AIR, DISEASES OF).

EUSTACHIAN TUBE, CATHETERIZATION OF (see EAR, EXAMINATION OF).

EVIDENCE (see MEDICAL EVIDENCE AND REPORTS ON MEDICO-LEGAL CASES).

EXERCISES, REMEDIAL (see REMEDIAL EXERCISES).

EXFOLIATIVE DERMATITIS (see DERMATITIS EXFOLIATIVA).

EXOPHTHALMIC GOITRE (*syn.* Graves's Disease; Basedow's Disease).—A disease due to an abnormal activity of the thyroid gland, the cause of which is unknown.

Etiology. Exciting causes.—The actual cause of Graves's disease is so far unknown, but it is clear that events which produce a marked impression on the emotions play an important part in the development of the disease. In many cases there is a history either of an accident or of a sudden shock, or of a prolonged period of mental strain or anxiety shortly before the onset of the symptoms. The strain of war is an important factor in both soldiers and civilians, as many cases occurred in Alsace and Lorraine after the Franco-Prussian War of 1870. During the Great War a considerable number of cases in soldiers came under my observation in military hospitals, and in these the strain of active service conditions, and not injury, appeared to be the chief factor.

Predisposing causes. Age.—The disease is rare in childhood and in old age. The great majority of cases occur between 15 and 50, and especially between 20 and 30 years of age. **Sex.**—Women are about ten times more liable to Graves's disease than men. **Heredity.**—Exophthalmic goitre may appear in two successive generations. Less rarely, two or more sisters have been affected, and other members of the family may suffer from different forms of thyroidal disease.

Pathology.—The thyroid gland shows certain characteristic changes. The alveoli are more numerous owing to the enlargement of the gland, and the secreting surface of many of them is increased by plication of the walls. The cells lining the acini of the gland are columnar instead of cubical, and the colloid secretion is changed in appearance and in

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staining reactions. The changes indicate that hypersecretion is taking place. In cases of long standing a considerable amount of interstitial fibrosis may be found. The total output of thyroidal hormones is increased, but it is possible that they are not present in the same proportions as in health. Other evidence that the symptoms of the malady are due to the action of an excess of thyroidal hormones is furnished by the clinical contrast between Graves's disease and myxedema, in which they are lacking. Direct evidence has also been provided on the one hand by the development of the symptoms of Graves's disease after the ingestion of excessive quantities of thyroid extract, and on the other by the disappearance of the malady after the reduction of the hypersecretion by removal or atrophy of the gland. The stimulating effect of the excess of thyroidal hormones may raise the basal metabolic rate by as much as 50 per cent. The exophthalmos is due to an accumulation of fat behind the eyeball, though vascular engorgement and spasmodic contraction of Müller's muscle have also been suggested as causes, on insufficient grounds.

Symptomatology. Onset.—The symptoms generally develop slowly, and some weeks or months of indifferent health may elapse before the nature of the illness becomes recognizable. A period of lassitude and easily induced fatigue may precede the development of any more definite symptoms. Progressive loss of weight with increased appetite may also occur early. Less commonly the onset may be sudden or acute, the characteristic symptoms appearing within a few hours or days. Continued or intermittent palpitation is in many cases the earliest definite symptom complained of. Enlargement of the thyroid gland is the first sign in some primary cases, and in all the cases in which the disease is secondary to a pre-existing simple goitre, a variety of the malady which has been described as toxic adenoma with hyperthyroidism.

The thyroid gland.—Some increase in the size of the thyroid gland is nearly always present; it comes on gradually during a period of several weeks or months. The enlargement is usually visible, but may only be detectable on careful palpation. As a rule it is moderate and uniform even in the fully developed stages of the malady. In a few cases the goitre reaches a considerable size, but it rarely causes any symptoms by mechanical pressure on the trachea or oesophagus. Once the dis-

ease is fully developed the size of the goitre only varies slightly at times, as during the menstrual period. Pulsation of the gland is often visible and is transmitted from the carotid arteries, although a true expansile pulsation may occur. A systolic thrill may be felt, and a murmur can be heard with the stethoscope over the gland in severe cases. In favourable cases, in the later stages of the disease the goitre may shrink and become firmer in consistence.

Ocular symptoms.—*Exophthalmos*, which is present in about 80 per cent. of the cases, is one of the most striking features of the disease (Fig. 27). It may vary from a very slight protrusion of the eyeball to extreme proptosis. In well-marked cases the eyeballs protrude so far that the sclerotic is clearly visible all round the margin of the cornea, and the eyelids are retracted and seldom move, so that a very startled or staring expression is given to the face and chronic conjunctivitis is often present. *Stellwag's sign* is the name given to the widening of the palpebral fissure caused by a spasmodic retraction of the upper eyelids. It may occur before there is any true exophthalmos, and in the earliest stage may only appear when the patient looks sharply to one side. *Von Graefe's sign* is due to delayed descent of the upper eyelid, so that when the patient looks downwards the upper eyelid is delayed for a brief period and the sclerotic becomes visible between the upper margin of the cornea and the eyelid. Weakness of convergence is sometimes present. A general weakness of the external muscles of the eye is not uncommon, and in rare cases there is total external ophthalmoplegia with ptosis. Sight is usually unaffected, and the reactions of the pupil are normal.

Circulatory system.—Increased frequency of the pulse is a constant symptom, and may be the first to appear. The frequency when at rest varies from 90 in mild cases, up to 140 or 160 in severe ones, 120 being the usual pulse-rate in the majority. The heart is irritable, so that slight exertion or emotion may cause an acceleration of 20 to 30 beats a minute. Throbbing of the carotid arteries and abdominal aorta is often complained of. The area and force of the cardiac impulse are increased, and may give the impression that the heart is enlarged. It is, however, only in advanced stages of the disease that actual hypertrophy and dilatation of the heart are found. The first sound is accentuated, and

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occasionally is audible several inches away from the chest-wall. In many cases a rough systolic murmur is heard over the pulmonary area. With dilatation of the left ventricle a mitral systolic murmur is often present, and in the later stages there may be oedema of the legs and other signs of heart failure.

Nervous system.—A condition of continual agitation and nervousness is present in nearly all cases. In some, restlessness is very marked. Many patients worry about trifles, and are fussy about details. Periods of depression are not uncommon. Actual insanity may develop, in the form either of melancholia or of mania. A fine regular tremor of the extended hands, at the rate of about eight movements a second, is present more or less in all cases, and in a few the tremor is coarse and jerky. Other functional disorders, such as a sudden giving way of the legs and *astasia-abasia*, occur.

Cutaneous system.—Brown pigmentation of the skin is often present, especially on the eyelids, the back of the neck, the axillæ, the areolæ of the nipples, and the flexor surfaces of the joints. The skin is warm and moist, and excessive local or general sweating is not uncommon. Any liability to chilblains disappears, but returns as the patient recovers. Sufferers from Graves's disease also remain remarkably free from "common colds," but this immunity diminishes as the patient improves. Owing to the moisture of the skin, its electrical resistance is diminished. Irregular subcutaneous swellings occur in some cases,

the true nature of which is uncertain. Loss of hair often occurs in the earlier stages of the disease, and in exceptional cases there may be complete alopecia. The nails may be thin and brittle, or opaque.

Digestive system.—In the early stages of the malady the appetite is often increased and there may be a constant craving for food. Increased frequency of defæcation up to three

or four times a day, without diarrhoea, occurs in many cases. In some there are recurring intestinal crises, characterized by painless diarrhoea of sudden onset and abrupt cessation, lasting for one or two days. The gastric crises or sudden attacks of vomiting which occur in severe cases are highly dangerous, as the vomiting may prove to be uncontrollable and the patient dies of exhaustion.

Genito-urinary system.—The urine is generally normal, but slight intermittent albuminuria is occasionally found. Glycosuria has frequently been observed, and true diabetes may develop. Menstruation is often

irregular, and amenorrhœa may persist for several months at a time. Sexual appetite may be increased.

Diagnosis.—In a fully developed case the diagnosis presents no difficulty. In slight cases, or in the early stages of the malady, its true nature is more easily overlooked, as the loss of weight, early fatigue, palpitation, and various subjective sensations may be attributed to other causes. A persistent increase of the pulse-frequency, in the absence of any



Fig. 27.—Exophthalmic goitre. (Dr. Murray's case.)

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fever or obvious cardiac disease, should always lead to a search for other signs of Graves's disease, such as tremor of the hands and an increase in size of the thyroid gland. It must also be remembered that all ocular signs are absent in one out of every four or five cases. Sometimes the enlargement of the thyroid is not obvious, and can only be detected on careful palpation. In a very few cases no palpable enlargement is present. In doubtful cases a rise in basal metabolism indicates that the symptoms are due to hyperthyroidism.

Complications and sequelæ.—Alimentary glycosuria is met with in some cases and indicates a low sugar tolerance, while severe diabetes may occur as a complication or may follow recovery from Graves's disease. Dilatation of the heart with dropsy is present in the late stages of some chronic cases which end by sudden or gradual heart failure. Acute exacerbations of all the symptoms or crises are sources of great danger. The pulse-rate increases up to 200 or more, there is free sweating, there may be vomiting, diarrhoea, great restlessness and even delirium. One or more of these symptoms may predominate; thus, gastric crises with persistent vomiting may cause death from exhaustion, or tachycardia with a pulse-rate of over 200 may be the chief symptom and end in cardiac failure. In a few cases, owing to progressive atrophy of the thyroid gland, recovery is followed by the development of myxœdema.

Prognosis.—The course of the disease varies greatly, but, speaking generally, in quite one-half of the cases either recovery takes place or the symptoms become so mild as to be negligible in the course of a few years. About one-quarter succumb to the malady or to one of its complications. In the remaining cases the disease is chronic, and the symptoms may persist for a number of years. The acute exacerbations or crises already mentioned are of ill omen. A very high pulse-frequency of 150–200 is a bad sign. In chronic cases gradual dilatation of the heart with œdema of the legs indicates cardiac-muscle failure and generally ends fatally.

Treatment. General.—In severe cases complete rest in bed is required until definite improvement has taken place. In those which are mild, rest for twelve hours in bed at night and for two hours on a couch in the afternoon may be recommended. If possible, most of the day should be spent in an open-air shelter. A dry, bracing inland climate is

to be preferred; in many cases residence at the seaside has proved to be harmful. The amount of walking exercise must be regulated according to the pulse-rate and general condition of the patient, but all active forms of exertion must be forbidden. If loss of weight has taken place, extra milk should be given in quantities of from two to five pints in the twenty-four hours until the normal weight is regained. The diet should consist chiefly of carbohydrates and fats with the exception of butter. Red meats, soup, and sea fish should either be omitted or given very sparingly.

Radiotherapeutics.—By the employment of X-rays or radium a direct attempt is made to inhibit and destroy the superabundant secretory cells in the thyroid gland and to induce the development of an interalveolar fibrosis, so as to diminish the functional over-activity of the gland.

The X-ray treatment of many of my cases has been carried out by Dr. A. E. Barclay, who employs the largest dose which can be given without injury to the skin. As only β and γ rays are required, α -rays are filtered out by passing them through a layer of 3 mm. of aluminium and a layer of boilerfelt on the skin. The usual dose is from one to one-and-a-half Sabouraud units measured on the tube side of the filter. A very hard tube is used. The treatment is given twice a week for the first few weeks, then once a week, and later at lengthening intervals. Even when all the symptoms have subsided it is advisable to give three or four more doses, at intervals of a month or more, to obviate any tendency to relapse. The danger of reducing the functional activity of the thyroid gland below the normal level and so causing myxœdema by too prolonged treatment is a remote one. Fortnightly or monthly estimations of the basal metabolic rate will give valuable indications of the progress of the case. The skin must be carefully watched, otherwise it may become permanently disfigured by telangiectases.

The radium treatment of my cases has been carried out at the Manchester Radium Institute, by Dr. A. Burrows, by the application of plates of a strength of 2.5 milligrammes or millicuries to the square centimetre over the enlarged thyroid gland. The plates were of such size that 45.0 to 76.5 mg. were applied each time, screened by 1.5 mm. of lead. The plates were applied for twenty-four hours and the application was repeated about every six

EXOPHTHALMIC GOITRE

weeks. As the primary effect may be to stimulate secretion, radium is unsuitable and even dangerous in acute cases.

Radiotherapeutic treatment may be continued up to twelve months, or even two years. In many cases good results have followed both methods of treatment, and they show that improvement and recovery in the majority of cases are accelerated by radiotherapy.

Electrotherapeutics.—When radiotherapeutic treatment is not available a mild faradic current may be used instead. Two flexible electrodes 4 in. by 2 in. are applied to the front and back of the neck and fixed by means of straps. A mild faradic current, just strong enough to produce a tingling sensation in the skin, is passed through the goitre from the secondary circuit of a dry-cell battery for one or two hours night and morning. The treatment may be continued for twelve months or longer with advantage.

Medicinal treatment.—Many drugs have been employed, but only a few have proved to be of real service. Arsenic is often of value, but it should be given in small doses over long periods; 3-5 min. of liquor arsenicalis may be given three times a day after meals. If the arsenic is omitted for a week every month it may be continued for six months. Belladonna has sometimes proved useful. Neutral sodium phosphate, 15-60 gr. three times a day, has also been used with benefit. The serum, blood, and dried milk of animals taken after thyroidectomy have been largely employed under the names of "antithyroidin," "thyroidectin," and "roda-gen" respectively, but no decisive results have been obtained. Of this class of preparation the fresh milk of a thyroidless goat has, on the whole, given the best results in this country. The use both of thymus and of suprarenal gland in the form of 5-gr. tablets three times a day has been followed by good results. With the exception of arsenic, I have, however, not found that any of these remedies give constant results. When anemia is present, iron should be combined with the arsenic. Tincture of convallaria, 10-15 min. three times a day, is helpful in some cases with marked tachycardia, but other cardiac tonics are not found to be of use unless there are signs of heart failure with dilatation of the ventricles, when digitalis and strophanthus find their opportunity. When restlessness and nervous agitation are pronounced, bromides give relief. Thyroid extract and all preparations contain-

ing iodine must be avoided, as they only aggravate the symptoms.

Certain urgent symptoms or crises require prompt treatment. In gastric crises the persistent vomiting should be treated by rest in bed, and rectal injections of half a pint of warm water, in which a drachm of sodium bicarbonate is dissolved, should be given every four hours. A hypodermic injection of morphia $\frac{1}{4}$ gr. should be given every eight hours. Pituitrin hypodermically has been followed by a cessation of the vomiting in some cases. During the sudden attacks of diarrhoea only milk should be allowed, and a mixture containing tinct. opii 5 min. with acid. sulph. dil. 15 min. may be given every four hours. Severe attacks of palpitation are relieved by the application of an ice-bag or a cloth saturated with evaporating lotion over the præcordium.

Surgical treatment.—In cases of secondary Graves's disease or toxic adenoma in which there are signs of pressure on the trachea, partial thyroidectomy should be performed without delay. In these cases, even when there are no pressure signs, surgical treatment is frequently required. In cases of primary Graves's disease, surgical treatment is rarely advisable until a full trial has been made, for six or twelve months, of the medical treatment already described. If no definite improvement has then taken place, surgical treatment must be considered. Two operations have proved to be valuable—ligature of the superior thyroid arteries, and partial thyroidectomy. Great improvement, but rarely complete recovery, follows the former operation, so that many surgeons prefer to perform a partial thyroidectomy at once. In order to obtain satisfactory results, not less than three-quarters of the enlarged gland should be removed. This may be done in two stages, one lobe being removed at the first operation, and part of the other lobe after an interval of several months. An operation is not advisable in early or slight cases. In severe cases an operation should not be undertaken, as a rule, if signs of myocardial failure or mental derangement are present, or during the occurrence of acute crises. A successful operation is often followed by rapid improvement, and in some cases by complete recovery. The disease, however, sometimes relapses within a year of the operation. Even in the most favourable circumstances the mortality from cardiac failure or toxic hyperthyroidism is not less than 5 per cent.

GEORGE R. MURRAY.

EXOSTOSES

EXOPHTHALMOS (*see* ORBIT, AFFECTIONS OF).

EXOSTOSES.—Non-malignant tumours composed of bone, which are sometimes called osteomata, and also certain bony outgrowths due to trauma or inflammation. True osteomata may be composed of either cancellous or compact bone.

CANCELLOUS OSTEOMATA

Etiology.—These osteomata are often hereditary; in such cases the tumours are multiple, with a more or less symmetrical distribution, and affect not only the flat bones and especially the scapulæ, but also the long bones. Their actual cause is unknown, but it has been suggested that they are the result of defective development of the periosteum near the epiphyses, whereby it lacks power to limit the lateral outgrowth of osteoblasts from the growing ends of the diaphyses.

Pathology.—A cancellous osteoma grows most commonly from the end of the diaphysis of a long bone, and forms a pedunculated tumour projecting obliquely among the surrounding tissues. It increases in size during adolescence and ceases to grow when the bone reaches full development. A bursa usually overlies it, and may communicate with an adjacent joint.

Symptoms are due to inflammation of the bursæ overlying the tumours and to pressure on adjacent structures, especially nerves and vessels. When large, these osteomata may interfere greatly with the movements of the limb and with its functions, and by their weight cause considerable discomfort.

Treatment.—If necessary, the tumour may be removed by dividing its neck with forceps and dissecting it away from neighbouring soft parts. In young people, osteomata have a cap of epiphyseal cartilage, and recurrence will ensue unless this is removed completely. With very large tumours, removal may be impracticable; if they cause much discomfort and render the limb useless, amputation may have to be considered.

Subungual exostoses, which cause a rounded swelling under the nail of the great toe and are very painful, should be treated by removing the nail, incising the nail-bed, and clipping the tumour away with cutting pliers.

Leontiasis ossea is considered in a separate article.

COMPACT OSTEOMATA

These are sessile tumours and grow from the compact tissue of certain of the skull bones on either their external or internal aspects. They originate most commonly in the frontal bones, the wall of the external auditory meatus, the antrum of Highmore and occasionally in other nasal sinuses, and in the jaws.

Symptoms.—Deformity and pressure symptoms are produced, the most serious being headache and other symptoms of cerebral compression if the tumour is intracranial. By the variety that grows in the ear gradually increasing deafness is caused.

Treatment.—The great density of the bone forming the tumours makes them difficult of removal unless this can be done by division of normal surrounding bone; special finely-tempered and electrically-driven bone instruments are necessary if the tumour itself has to be cut.

INFLAMMATORY EXOSTOSES

Exostoses produced by inflammation occur in certain situations as a result of small traumata, or the detachment of some of the fibres of a fascial origin. "Rider's bone" is an ossification of the upper part of the adductors; there is a bony projection on the inner part of the thigh, capped by an adventitious bursa which is liable to attacks of inflammation that cause pain on pressure. If it is troublesome it must be chiselled away.

Another not uncommon form of inflammatory exostosis is a small spur of bone growing from the under aspect of the os calcis at the attachment of the plantar fascia; there is an overlying bursa, and pain is produced by walking on uneven ground or any cause of trauma. On examination there is tenderness on pressure, and slight thickening of the heel under the exostosis can be made out. All symptoms are as a rule immediately relieved by cutting a small depression in the inside of the heel of the boot at the spot which corresponds with the lesion; occasionally operation is indicated, the bony outgrowth being exposed and chiselled away through an internal incision.

C. W. GORDON BRYAN.

EXTERNAL-RECTUS PALSY (*see* OPTHALMOPLÉGIA).

EXTRA-SYSTOLES (*see* HEART-BEAT, ABNORMALITIES OF).

EXTRA-UTERINE GESTATION (*see* PREGNANCY, EXTRA-UTERINE).

EYE, CONGENITAL ANOMALIES OF

EXTRAVASATION OF URINE (*see URINE, EXTRAVASATION OF*).

EXUDATES, EXAMINATION OF (*see BACTERIOLOGY AND PATHOLOGY, CLINICAL*).

EYE, CONGENITAL ANOMALIES OF.

—The subject of congenital abnormalities of the eye is one of great complexity, and little more can be attempted here than to indicate in general terms how they originate. In normal development the optic nerve, the retina, and the pigment epithelium lining the choroid, iris, and ciliary body are the product of a hollow outgrowth from the epiblastic cerebral vesicle. The lens is formed by a hollow downgrowth from the surface epiblast. This "lens vesicle," meeting the "optic vesicle" somewhat below its apex, invaginates it, as one might indent a soft rubber ball, so as to produce a groove on its lower aspect (the fetal cleft). Mesoblast surrounds both structures and passes through the fetal cleft into the space between the lens and the optic vesicle (primitive vitreous). The optic vesicle now grows round and encloses the lens vesicle (leaving only the pupillary opening in front), and the edges of the fetal cleft meet and fuse so that all traces of it are obliterated. From the surrounding mesoblast the cornea, uveal tract, and sclera are developed, and during fetal life an artery (the hyaloid artery) passes forward from the optic disc to the back of the lens, where it spreads out to enclose the lens in a "fibro-vascular capsule," from which the venous blood is drained through the vessels of the iris into the anterior ciliary veins; this vascular apparatus normally disappears before birth.

Almost all congenital malformations of the eye result from **the persistence of mesoblast beyond the normal period of its existence**. Roughly speaking, there are three possibilities:

1. The mesoblast may **persist permanently**, the fetal cleft remaining patent, or not closing in the normal manner. Owing to the presence of an abnormal strand of fibrous tissue the eye remains small and undeveloped, and usually shows a number of malformations (*microphthalmos*). If the gap in the cleft is wide the retina may grow through it into the orbital tissues, where it becomes degenerate and cystic (*microphthalmos with cyst*).

2. The mesoblast may **persist long enough to prevent normal closure of the cleft, yet finally disappear**. In this case the mesoblast

in the line of the cleft fails to differentiate properly into uvea and sclera, and the resulting "*coloboma*" may occur in any part of the normal extent of the cleft, i.e. in the lower part of the eye anywhere from the optic disc to the pupillary margin. *Coloboma of the choroid* takes the form of a large gleaming white parabola-shaped area, traversed by branches of the posterior ciliary arteries; the area often becomes ectatic under the intra-ocular pressure. *Coloboma of the ciliary body* cannot usually be detected by clinical methods. In *coloboma of the iris* a complete gap is present below, and the pupil has the figure of a key-hole or of an inverted Gothic arch. The iris is also sometimes almost totally undeveloped (*aniridia*), a condition which not infrequently leads to glaucoma. *Coloboma of the lens*—a nick in its equator—is occasionally observed.

3. The mesoblast may **persist permanently yet not impede the closure of the cleft**. In this case it is the mesoblast in the centre of the vitreous which remains. A strand connected with the arteries of the disc is termed a "*persistent hyaloid artery*," though it seldom contains a patent vessel. A portion of the same material adhering to the back of the lens is called a *posterior polar cataract*. Remains of the connexion between the anterior portion of the fibro-vascular capsule and the vessels of the iris give rise to a *persistent pupillary membrane*—fine strands crossing the pupil and uniting, not with the pupillary margin, but with the vascular arches in its vicinity.

Abnormalities unconnected with the intra-ocular mesoblast.—Among this group of abnormalities are *coloboma* and adhesion of the lid to the globe (*symblepharon*). *Congenital ptosis* is usually bilateral, and the condition tends to run in families. The defective action of the levator palpebræ is reinforced so far as possible by overaction of the frontalis, so that the skin of the brow is thrown into wrinkles, and the eyebrows are raised. In order to see under the drooping eyelids the patient throws his head back, a highly characteristic attitude being thus assumed. There is usually an associated defect of the superior rectus. Operation is imperatively indicated when the lid covers the pupil; in other cases it may be undertaken for cosmetic reasons, or various mechanical devices for raising the lid may be tried.

A difference in the colour of the two irides (*heterochromia*) may be a congenital abnormality or due to chronic iridocyclitis. Before

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deciding on the former alternative the lighter-coloured eye should be carefully examined for cellular deposits on the back of the cornea (keratitis punctata).

Opaque nerve-fibres appear as dense white areas with feathery borders, situated at the edge of the disc.

GEORGE COATS.

EYE, DISEASES OF (see AMAUROSIS; AMBLYOPIA; CATARACT; COLOUR VISION; CONJUNCTIVITIS; CORNEA, AFFECTIONS OF; DAY BLINDNESS AND NIGHT BLINDNESS; GLAUCOMA; HEMIANOPIA; OPHTHALMOPLEGIA; OPTIC ATROPHY; RETINA, AFFECTIONS OF; SCLERA, AFFECTIONS OF; SCOTOMA; UVEAL TRACT, AFFECTIONS OF; VITREOUS, AFFECTIONS OF).

EYE, EXAMINATION OF.—The examination of the eyes must be conducted in a very methodical and careful manner, since mistakes in diagnosis are usually the result of imperfect knowledge of the guiding principles, and some points are often so minute that unless they are looked for definitely they may be entirely overlooked.

For this purpose the general practitioner must possess a certain equipment in his consulting-room, which, however, neither involves a serious outlay in expenditure nor takes up an undue amount of space. The following are the most essential:—

A Snellen's test-type card arranged for 6 metres and 5 metres distance, as some rooms are not long enough to accommodate a 6-metre range.

A box containing convex and concave lenses of varying strengths up to about 12 D. These need not be numerous, as the general practitioner does not usually require to prescribe glasses; if he does, a larger box must be procured.

An ophthalmoscope with two large mirrors (plane and concave), in addition to a small concave tilted one.

A +13 D lens, generally sold with the ophthalmoscope, preferably with a handle.

A pocket lens of 12 diameters magnifying power.

Small bottles of fluorescein 2 per cent. and cocaine 3 or 4 per cent. (to relieve pain when making an examination of the cornea).

A well-arranged electric lamp with frosted glass.

Glass rods, cotton-wool, etc.

With the assistance of this equipment and a well educated use of the hands and eyes, the practitioner is in a fair way to make a sufficiently accurate diagnosis in most diseases of the eye.

Two separate methods of examination are required, one in cases where there is some definite indication pointing to the particular part of the eye to be examined, and the other where there is no external manifestation to explain the cause of the symptoms complained of. These will be described separately.

1. EXTERNAL EXAMINATION

Supra-orbital ridge.—Pass the finger along the ridge and feel for any unevenness or fullness between it and the eyeball; there ought to be an appreciable depression between this ridge and the globe, into which the tip of the finger can be inserted, and in which can be felt any swellings that may be protruding from behind the eye or from the roof of the orbit or ethmoidal sinuses. Always compare the two sides; any difference in the prominence of the frontal regions should be noted.

Proptosis.—Any suspected prominence of the eyeball should be examined for in the following way: Stand behind the patient, who should be seated on a chair and looking straight in front of him, and raise the upper lids, holding them against the supra-orbital ridge with the forefinger of each hand. Then arrange the point of view so that the cornea is just visible beyond the line of the supra-orbital ridge. Next, without moving the head (observer's), compare the position of the cornea in each eye in relation to the supra-orbital ridge, when the slightest prominence can at once be detected. Many eyes which appear to protrude, when looked at from the front, fail to pass this test. Also notice the direction of the proptosis, i.e. directly forwards, or to one side or the other.

The presence of **enophthalmos** is shown by placing the edge of a card so that it touches the malar bone below and the supra-orbital ridge above, and comparing the distance of the cornea from the edge of the card in the two eyes.

The **condition of the orbit** can be examined by noting the presence or absence of proptosis and other signs, and the cavity can be explored to a certain extent, by passing the finger above between the orbital ridge and eyeball as far back as possible, and also between the lids and the globe. In order to

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facilitate the latter examination the eye must be cocaineized.

Diseases of the orbit are characterized by four signs, which differ in severity and in their relationship to each other according to the disease that is present. They are: (1) Proptosis, (2) limitation of movement, (3) cedema of the conjunctiva and dilatation of its blood-vessels, and (4) the condition of the fundus, especially of the optic nerve.

For example, in orbital cellulitis, where the inflammatory exudate occupies the whole of the orbit, we notice well-marked proptosis, chemosis due to obstruction of the vascular and lymph channels, and limitation of movement due to interference with the action of the muscles in the orbital cavity, but the optic nerve is unaffected. On the other hand, tumours of the optic nerve do not interfere with the muscular action nor with the vascular and lymph flow, and therefore the leading signs are proptosis and optic neuritis. Similarly, we can apply these signs to other conditions of the orbit. The direction of the proptosis is important, as is also the situation of the part where the limitation of movement is most manifest.

The size and position of the **palpebral aperture** should be noticed, and whether the eye can be covered properly by the lids when they are closed, failure in which indicates some imperfection in the action of the orbicularis. It should also be noticed whether there is any drooping of the upper lid (ptosis), which may be congenital or an early sign of some nerve disorder.

Inflammations of the **lacrimal gland** are diagnosed by the presence of a tender swelling at the outer margin of the orbit between it and the globe, and obliterating the space beneath the supra-orbital ridge; the swelling is particularly tender, quite out of proportion to its size, and generally the preauricular gland is also enlarged.

The **lids** should be examined first on their skin surface; any convexity of the lids forwards indicates longstanding disease on the conjunctival surface which has led to contraction of the tarsal plate and folding-in of the contracted tissue. If the lash-bearing edge of the lid is red and swollen there is blepharitis due to infection of the sebaceous glands surrounding the hair-follicles. Some cases of intense irritation of the eyeball, either corneal or conjunctival, may arise from ingrowing eyelashes; this is obvious enough when the whole line of the lashes is turned in, but not so apparent

when only one or two are growing inwards, and a careful examination must be made with the pocket magnifying lens or a binocular loupe.

The inner or **conjunctival edge** of the lids should be examined for any indication of commencing inflammation of the Meibomian glands. Abnormal activity of these glands causes a white frothy secretion which sticks to the edges of the lids, and often a good deal of irritation, but it must not be confused with the secretion found in conjunctival inflammations.

The **lids** should be everted in order to examine their conjunctival surface. This is easy in the case of the lower lid, the patient being directed to look up while the lid is pulled down. To evert the upper lid is less simple. Two methods may be employed, one in which both hands are used, and the other by means of one hand only. For both methods it is essential that the patient should look well down; it is impossible to carry out the movement otherwise. If two hands are used, stand in front of the patient, place the thumb (the right in the case of the left eye, and the left in the case of the right eye) just on the margin of the orbit, the tip projecting downwards so as to touch the lid near the lashes, and push slightly backwards; then, with the forefinger and thumb of the other hand, take hold of the lashes and pull forwards and upwards against the thumb already in position, at the same time pressing slightly backwards with the latter. In this method, instead of the thumb a glass rod or pencil may be used and placed on the upper lid parallel to the margin. To evert the upper lid with one hand, use the right hand for the left eye and the left hand for the right eye. Place the tip of the forefinger on the skin surface of the upper lid near the outer canthus, and the thumb close to the lash-bearing edge of the lower lid, and then push the lower lid upwards until it is just underneath the edge of the upper one; draw the forefinger already in contact with the upper lid slightly outwards, at the same time pressing backwards, when it will be found that the thumb can easily be transferred from the lower lid to the under surface of the upper. The upper lid is now held at its edge between the thumb and forefinger, and with a slight backward and rotatory movement the lid can be rolled over the end of the forefinger. Thus the conjunctival surface of the tarsus is exposed.

To see the upper fornix, by far the best way is, while the fingers are holding the upper lid

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everted, to place the forefinger and middle finger of the other hand against the lower lid near the lashes and push it against the globe, which is thus thrust slightly backwards into the orbit, when the upper fornix comes into view without any difficulty.

The **lachrymal passages** are investigated by noting first the size of the punctum, whether it is properly applied to the globe so that it points upwards and backwards, and also the patency of the punctum, canaliculus, sac, and duct. To ascertain the patency of the duct, place a drop of fluorescein inside the lower lid, and after a few moments ask the patient to blow his nose; if the duct is patent a stain will be found on the handkerchief. Another way is to inject some plain water into the duct with a very fine lachrymal syringe specially made for the purpose; any diminution in the size of the punctum will be noticed at once by the difficulty felt in introducing the nozzle, but unless cicatricially contracted it may be dilated with a cone-shaped probe (Nettleship's). If the duct is open, fluid ought to pass down into the nose and thus to the pharynx, when the patient will make swallowing movements.

The **caruncle** can easily be seen and examined; a pocket magnifying lens is useful for the examination. Sometimes hairs will be found scratching it, especially when, as occasionally happens, a hair becomes impacted in the upper or lower punctum. When this occurs, localized redness will be noticed on the adjoining part of the ocular conjunctiva, whilst all other parts are normal.

The conjunctiva of the lids has already been dealt with, but the **ocular conjunctiva** must also be carefully examined. Note whether there are elevations in any part, e.g. lymphatic cysts, dermoid swellings, pinguicula or pterygium, the last two being always found on the part exposed between the lids; whether conjunctival injection is general or localized, and if the latter, whether it is near the limbus or only at the angles. In some conditions the conjunctiva is oedematous, exhibiting a shiny, jelly-like appearance which can be rendered more evident by gently pushing the edge of the lower lid up against the globe. This is known as *chemosis*.

The **sclerotic** must be examined for any injection, swelling, or thinning. Injection indicates inflammation and is usually situated near the limbus (sclero-corneal margin); swelling may be localized or occur all round the limbus, and thinning is shown by a dark

discoloration just behind the limbus, due to the choroidal pigment showing through the attenuated sclera.

Ciliary injection is a sign with which every practitioner must make himself thoroughly familiar, as it is common to so many conditions of extreme importance. It consists in a dilatation of the ciliary anastomosis of the deep vessels surrounding the limbus, and is a sign of inflammation of the cornea, iris, or ciliary body, or perhaps of all of them together; it points to the necessity for the use of atropine. It is true that it occurs sometimes in cases of acute glaucoma, when atropine is unsafe, but in these circumstances there are other signs and symptoms which confirm the diagnosis.

Sometimes ciliary injection exists simultaneously with conjunctivitis, but in this case it will be noticed that there is a distinct area on the globe between the enlarged conjunctival and ciliary vessels where a relatively white colour is visible. The conjunctiva containing the dilated vessels can always be moved on the globe beneath, whereas ciliary injection is not affected by this manoeuvre.

The cornea.—Whenever pricking pain or photophobia is complained of, the cornea must be carefully examined in the three following ways:—

1. **For loss of brilliancy.**—Let the patient stand opposite a window, when an image of the window will be reflected on the cornea. Then instruct him to follow the movements of an object (e.g. a finger) through different parts of the field of vision. In this way the image of the window is thrown on to different parts of the cornea, and the observer can notice whether it is equally bright at all points on the surface; any part showing a duller reflex than another indicates some defect of the cornea at that point.

2. **Staining.**—Apply a drop of 2-per-cent. fluorescein from a glass rod to the upper part of the eyeball not far from the limbus, while the patient is looking down. Follow this by a drop of water (if there is no photophobia) or 2-per-cent. cocaine (if there is photophobia), and then notice if that part where the dullness was observed shows any greenish-yellow staining. If this is present there is ulceration or an abrasion; if not, the corneal epithelium is intact.

3. **Examine with a pocket magnifying lens** under oblique illumination to see the nature of the dullness, and whether there are any additional spots, vessels, linear markings, or details to assist in the diagnosis. Spots on the cornea

EYE, EXAMINATION OF

may be keratitis punctata ; lines may indicate striate keratitis, seen in acute deep inflammations ; vessels may be deep or superficial, indicating the nature of the infiltration.

If there is any difficulty in opening the lids, as in cases of intense photophobia, lid-retractors must be used to avoid any pressure on the cornea, or cocaine may be instilled temporarily to relieve the pain. An ulcer may be on the point of perforation, which might easily be precipitated by any careless use of the fingers.

The iris.—This is a part of the eye which the most inexperienced can study with comparative ease. If once the general practitioner can make himself familiar with the various methods of examination of the iris, he will have a valuable guide to the diagnosis of many otherwise obscure cases in ophthalmology. The investigation can be carried out without the aid of any special instruments. It is one of the first steps in any ophthalmic examination, and from a general practitioner's point of view is perhaps the most important of all.

Of the observations to be made in connexion with the iris, the following may be specially mentioned :—

1. The **reaction of the pupils to light** should be recorded in every case and conducted separately on each eye. Normally each eye should act both directly and consensually to light and also to accommodation. Let the patient stand opposite a window, or facing some light if daylight is not available, and cover completely the eye *not* under examination so as to exclude all light ; the direct action of the iris of the uncovered eye can then be tested by alternately shading and exposing it. A brisk contraction ought to take place whenever light is admitted. Next, while the eye to be tested is still exposed, uncover the opposite eye, when a further slight contraction to light will be observed, indicating normal consensual reaction. Carry out the same method of examination in the other eye. Then apply the test for accommodation with both eyes open by instructing the patient to look first into the distance and then at an object held about a foot away from the face. Contraction of both pupils ought to occur on fixing the near object. If these tests are properly conducted, variations in the different reactions may be noticed in abnormal cases.

(a) *No direct action but only consensual*—an indication of defective light-conducting mechanism in the eye under examination in some part

of the sensory tract ; i.e. the retina, the optic nerve, or the optic tracts.

(b) *No reaction consensually but only directly.*—This indicates defective light-conduction in the opposite eye, which may be verified by testing the direct action of the latter.

(c) *Neither direct nor consensual reaction.*—Unless the pupil is mechanically prevented from acting by iritic adhesion to the anterior capsule of the lens (i.e. old iritis), this represents the fixed dilated pupil seen in glaucoma and drug mydriasis, and shows either that there is third-nerve paralysis on the side under examination or that the light-conducting elements of the same or both sides are defective.

(d) *The Argyll-Robertson pupil*—action to accommodation but not to light.

(e) *Failure of the iris to maintain contraction* when light is thrown into and fixed on the eye by means of oblique illumination with a strong convex lens. Normally, after a few oscillatory movements of the iris which are normal (if exaggerated, sometimes called *hippus*), the pupil remains in a state of contraction ; if it does not, but tends to become dilated in spite of the continued presence of the light, this indicates commencing defects in the conducting elements of the eye under examination, and is a warning of the onset or existence of optic atrophy, retrobulbar neuritis, glaucoma, degeneration or detachment of the retina.

2. **Inequality of the pupils** is of extreme importance, since it must always be regarded as of pathological significance rather than of congenital origin, until a careful examination has been made. It is often difficult at first to determine whether the larger or the smaller pupil belongs to the affected eye ; this can only be settled definitely by reference to their reaction, and by a critical examination of the pupillary border by means of a pocket magnifying lens to discover the presence or absence of iritic adhesions. Speaking generally, dilatation means either paralysis of the third nerve, defect in the retina or optic nerve, or irritation of the cervical sympathetic, and contraction miosis apart from adhesions indicates either paralysis of the sympathetic or irritation of the third nerve. Inequality of the pupils, like failure of maintenance of contraction to light, may be the first sign of optic atrophy, glaucoma, or other defect of the conducting elements.

3. **The shape of the pupillary border.**—This must be noted, and observation made as to its regularity or position. If irregular, the probability is that old iritic adhesions have united

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the free margin of the iris to the anterior capsule of the lens in certain places (*posterior synechia*); this can be verified by instillation of atropine or by the magnifying lens. A pear-shaped pupil is often an indication of a tendency to or the existence of glaucoma, and a similarly-shaped pupil with the pointed end fixed while the rest of the pupil acts, especially if accompanied by shallowness of the anterior chamber opposite the point, indicates adhesion of the iris to the back of the cornea (*anterior synechia*).

4. **The colour of the iris.**—Difference in the colour of the iris, as compared with the other eye, is more likely to be pathological than congenital. The lighter-coloured iris (due to disappearance of the stroma pigment) is usually on the affected side, and generally the result of longstanding irido-cyclitis, so that cases presenting this peculiarity must be examined carefully.

A semi-translucent appearance often accompanies glioma of the retina and also occurs with some acute choroidal inflammatory deposits. Discoloration (or so-called muddiness) with loss of pattern, especially if associated with ciliary injection and fixed contracted pupil, is a sign of acute iritis.

5. **The actual size of the pupil**, apart from inequality, is important, since glaucoma may give rise to dilatation of the pupil on both sides, while small pupils are a common accompaniment of old age, and are physiological.

6. **Gaps in the iris or nodules on its surface** are either of congenital origin or associated with some pathological condition of the iris.

7. **Spots of iris pigment** on the anterior capsule of the lens, seen with the pocket lens, indicate old iritis.

8. **The depth of the anterior chamber**, i.e. the distance between the iris and the back of the cornea, must be noted, as it varies in pathological conditions as well as physiologically. The anterior chamber may be obliterated altogether, when the iris lies in contact with the back of the cornea: if this is accompanied by increased tension it indicates that the iris is adherent along its whole anterior surface; if accompanied by lowered tension it shows that the aqueous fluid is leaking away through some perforation or wound. A uniformly shallow anterior chamber is a common sign in glaucoma but may be physiological in old people; a deep anterior chamber probably indicates myopia, old irido-cyclitis, or dislocation or absence of the lens.

In dislocation of the lens the iris is also tremulous when the eye is moved in different directions, because it has lost its normal support. The chamber may be shallow at the circumference and deep in the centre—a condition seen in iris bombé, when the whole of the pupillary border is bound down to the anterior capsule of the lens so that the aqueous, being unable to escape into the anterior chamber, bulges the iris forwards. The anterior chamber may be shallow or deep on one side and normal on the other; the former indicates adhesions of the iris to the back of the cornea, and the latter a partial dislocation of the lens.

The lens.—Examination of the lens must always be carried out in a dark room by reflected light, for no observation is complete if made by oblique illumination alone, and is apt to be deceptive. A snow-white reflex behind the pupil indicates probably a mature cataract, but a distinctly grey reflex is common in sclerosed and old lenses, and, although simulating a cataract, is quite compatible with a perfectly clear lens. Take the patient into the dark room and arrange the light somewhere behind the head; then with the large concave mirror of the ophthalmoscope throw light into the suspected eye, the observer's eye being behind the sight-hole. In the normal eye a perfectly uniform red reflex, due to light reflected from the fundus, is noticed illuminating the pupillary opening. Any opacities in the lens will show up as black streaks or spots against this red reflex, and with a complete cataract there will be no red reflex at all. This can be verified by comparison with the other eye. A lens which is merely sclerosed will give a good red reflex. Opacities in the lens can also be observed by means of the direct method of ophthalmoscopy with a +12 D lens behind the sight-hole.

The vitreous.—This is examined in the same way as the lens, but in this case the opacities are movable and can be seen floating across the field of vision when the eye is moved in various directions and then brought to a standstill. Another method is by direct ophthalmoscopy and dull illumination, with anything from a +6 D to a +10 D lens behind the sight-hole while the opacities are set in motion.

II. CASES WITHOUT EXTERNAL INDICATIONS

We now come to the method of examination to be conducted in cases where there is nothing visible to the unaided eye to offer guidance in locating the part of the eye affected, the only

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symptom being defective vision in one or both eyes.

For this purpose a systematic routine should rigidly and patiently be carried through in every case, no matter what statements the patient himself may make regarding his own symptoms; they may be helpful or not, but, having carefully noted them, we must proceed with our examination and be deterred by nothing from carrying out every detail.

1. **Record the vision** in each eye separately in terms that everyone can understand by means of Snellen's test-types at a distance of 6 metres (20 ft.), and make a note of the lowest line that can be read at this distance, e.g. $\frac{6}{24}$, $\frac{6}{18}$, $\frac{6}{12}$. The distance at which the letters ought to be read is marked on the card; the numerator will represent the distance from the type that the patient is standing, and the denominator the distance at which the line in question ought to be read normally. If the patient is unable to read the top letter at any distance (e.g. 3 metres = $\frac{3}{60}$), or close up to the board (= $\frac{3}{30}$), then hold up fingers about 2 ft. away and gradually bring the hand nearer until he can count the number held up. If he is unable to see them at all, try moving the hand backwards and forwards between him and the light and see whether he can detect "hand movements." If he cannot recognize the hand moving, reflect light into the eye in a dark room from the ophthalmoscope mirror and notice if there is "perception of light" or not.

2. **Take the pupil reaction**, as described above (p. 469).

3. **Feel the tension**.—In clinical work we must rely on the sense of touch, and, since the tension of different eyes varies within certain physiological limits, it is all the more important to practise the tactile sensation as often as possible. For this purpose, direct the patient to look down, as no record of tension is of value unless the eyes assume this position; then place the forefingers close together on the upper part of the globe as far back as can conveniently be reached, and test the tension as gently as one would feel for fluctuation, at the same time keeping the hands steady by resting the remaining fingers on the forehead. Then compare the tension in the two eyes.

4. **Retinoscopy** (if necessary, under a mydriatic).—The case may be one of refractive error or intra-ocular disease, and we must always assume it to be the former until we have proved the contrary. The practitioner may not be able to measure accurately the refractive error,

for this requires many years of constant practice, but if he can recognize the shadows (as described under **REFRACTION AND ACCOMMODATION, ERRORS OF**), and can approximately estimate the glass which corrects the error, he is in a position to see whether any considerable improvement is possible with a correcting glass. He may not succeed in getting his patient to read $\frac{6}{12}$, but if he can improve the vision from say $\frac{6}{60}$ to $\frac{6}{12}$ he will make an apparently serious case appear far less formidable, and has advanced more than half-way towards making a diagnosis.

5. **Use of the ophthalmoscope**.—The first essential of ophthalmoscopic examination is to **examine the red reflex**. Take the patient into the dark room and let him be seated with his back to the light; then throw the light into the eye, as described in the paragraph on examination of the lens (p. 470). Apart from the opacities in the media already alluded to, this red reflex illuminating the pupillary area should be of the same intensity, no matter from what part of the fundus the light is reflected. If in any part of the fundus we observe, for example, a white, grey, yellow, or black reflex, instead of the normal red colour, we know that there is some abnormality in the interior of the eyeball—perhaps a new growth, an inflammatory deposit in the vitreous, choroid, or retina, or a detachment. The details may require to be settled by direct examination, but at least we have located the part of the eye where the cause of the bad vision is to be found.

The second step is to examine the fundus by the **indirect method**. With the patient seated in the same position as before, place the lamp on the right side on a level with the back of his head and about as high as the right ear and 3 or 4 in. away from it. Sit down straight in front of the patient. Use the large concave mirror for reflecting the light, place a + 2 D lens behind the sight-hole, hold the ophthalmoscope in the right hand, at the same time raising the little finger into a vertical position, and then proceed to examine the *right* eye. Ask the patient to look at the raised little finger, which brings the disc into a straight line with the sight-hole. To bring the image into focus, it is now only necessary to place the + 13 D lens held in the left hand opposite the eye and draw it *slowly* backwards and forwards, or a little from one side to the other, by making *very slight* movements, until the disc is plainly seen; the + 2 D behind the sight-hole keeps it in focus, and by *asking the*

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patient to look in different directions all parts of the fundus can be examined. Remember that no other movement must be made except by the left hand, which holds the +13 D lens.

To examine the *left* eye the same directions can be followed, only substituting left for right in the above description; but if the practitioner prefers not to change hands he can ask the patient to look at his (the practitioner's) left ear, when the left disc will come into view.

The indirect is the best method to get a general idea of the fundus condition, and also when the media are hazy and in cases of high myopia.

The third step is the examination by the **direct method**, by which the magnification is much greater; it is used for the investigation of all details.

The practitioner must examine the right eye with his right eye, and the left with his left.

For the *right* eye, place the lamp in the same position as for the indirect method and stand on the right side of the patient. Hold the ophthalmoscope flat on the hand with all the mirrors upturned, and rotate the mirrors so that the small concave tilted mirror is opposite the sight-hole; turn it with its inclination towards the right with the plain opening in the rotating disc of lenses opposite the sight-hole; the ophthalmoscope is now ready for use. Hold the ophthalmoscope *exactly vertically* in front of the eye about 2 or 3 in. away, and the light from the small mirror will shine on the cornea; then, by bending over, bring the eye opposite the sight-hole, and move the head, together with the ophthalmoscope in position, slightly backwards or forwards till the details of the fundus come into view. There may be some difficulty, while executing this manoeuvre, in keeping the light on the eye: if the red reflex is visible, one may be sure that the light is in the right position: but if not, the light is not being reflected into the eye. The patient at the time must be looking straight in front of him into the distance in order to relax his accommodation, and the practitioner must also learn to relax his own accommodation, otherwise no view of the fundus is obtained.

Accuracy in the positions of both patient and surgeon is essential to the direct method of examination, and a sharp look-out must be kept on anything likely to obscure the passage

of the rays of light from the lamp to the ophthalmoscope.

The *left* eye is examined in the same way, only substituting left for right in all the details described above.

6. If hitherto all examination has failed to reveal any obvious cause for the defective sight, the next step is **to test the field of vision**. The usual method is by means of a perimeter, by which the field can be recorded on a chart; but if no perimeter is available it can be tested with a fair degree of accuracy by mounting a small piece of white card, 10 mm. square, on the end of some suitable holder, and, while the patient with his back to the light stands opposite the practitioner, carrying the test object through various parts of the field. The field must be taken in each eye separately, and during the examination of the right eye the left must be closed and the patient directed to look into the practitioner's left eye; the visual field can then be compared with the normal.

Contraction of the field is met with in optic atrophy, glaucoma, etc.

7. Another valuable test is to examine for a **colour scotoma** in the central part of the field. This is carried out with a small coloured square on the end of a holder. Note whether the colour is seen more sharply and brightly in the central part of the field or a little way from the centre; if the latter, it indicates a central (partial or complete) scotoma for colour – a sign peculiar to inflammation of the papillo-macular bundle of nerve-fibres behind the globe, e.g. tobacco amblyopia.

8. The only remaining test is that of the **perception of light**. In all cases where a view of the fundus is unobtainable, owing to opacities in the media, or to organized exudate blocking the pupil, etc., reflect the light into the eye from various points in the field of vision, and ask the patient to indicate from which direction the light comes. If he is able to locate the position accurately, the retina is probably quite healthy; if not, it is insensitive, and incapable of perceiving an image, even though the obstruction to vision be removed by operation. No opacity is dense enough to exclude light, and, provided the retina is healthy, the perception of light sensation is as acute as when the media are all transparent.

MALCOLM L. HEPBURN.

EYE, FOREIGN BODIES IN (see EYE, INJURIES TO).

EYE, INJURIES TO :

EYE, INJURIES TO.—Injuries to the eye are of three varieties :

1. Foreign bodies, burns, irritating fluids or gases, leading to superficial damage to the cornea or conjunctiva, or both, without penetrating the globe.
2. Penetrating wounds, with or without retention of the foreign body inside the globe.
3. Blows on the eye, causing (a) complete rupture of the globe with a conjunctival wound, or (b) subconjunctival rupture of the globe. Either of these may occur with or without extrusion of the contents of the eye.

1. The commonest class of superficial injury to the eye which the practitioner is called upon to treat is that due to a **foreign body in the cornea or under the upper lid**. The latter causes more distressing symptoms than the former and provokes much pain and injection of the globe; but it is easily removed by everting the lid (*see* EYE, EXAMINATION OF), and rubbing the foreign body off with a small piece of moist cotton-wool, when immediate relief is experienced. A foreign body in the cornea demands much more care both in diagnosis and in treatment. Although the cornea is far more sensitive than the conjunctiva, a foreign body in it is more likely to be disregarded by the patient; he may not seek advice for two or three days, whereas one with a foreign body under the upper lid will come immediately for treatment. There is often no sign of irritation or inflammation of the eyeball. The foreign bodies likely to lodge on the cornea are particles from rapidly moving objects, such as a grindstone or a lathe. Many of them are hot and get embedded in the cornea beyond the nerve-endings, and thus do not cause much trouble when they have once become fixed. The patient often complains merely of a pricking sensation, which he regards as coming from beneath the upper lid. He is anxious to point out where it is situated, and thus may unintentionally deceive the practitioner, who, if the foreign body is small and has produced no sign of inflammation, may omit to make a careful examination of the cornea with a magnifying lens, and overlook it. If nothing is discernible, the diagnosis may be verified by staining with fluorescein, when the smallest foreign body will sometimes be mapped out with a ring of stain where the epithelium is

denuded, or an ulcer at the site of the foreign body may be seen.

Treatment.—Several methods of removal can be employed, in all of which care must be taken to disturb the corneal epithelium as little as possible. When normal epithelium has been shed, although the breach quickly heals if the eye be tied up, the new epithelium is insecurely attached and easily scratched off again, so that a recurrent abrasion of the cornea over the site of some former injury is very common. Infected foreign bodies may give rise to hypopyon ulcer.

If the foreign body is only just attached to the surface, a piece of moist cotton-wool screwed up into a point, or a sharp piece of card, is often sufficient to remove it without disturbing the epithelium; this method should always be tried first. Should this not succeed, an attempt must be made to lift off the foreign body with a blunt spud, but most are so deeply embedded that they have to be dug out carefully with a sharp needle; if any rust stain remains in the corneal tissue afterwards, it must be scraped away thoroughly at the same time, lest it produce irritation later. A corneal magnifying lens is always necessary to make sure that the foreign body is properly removed.

Another common injury is an **abrasion of the corneal epithelium** from a scratch by a finger-nail, hatpin, or similar object. It is often very difficult to see, but the sharp pricking pain, with much photophobia, ciliary injection, and lachrymation, shows that the cornea is damaged, and the diagnosis can easily be established by the application of fluorescein. **Treatment** consists in bandaging up the eye for twenty-four hours, washing out once or twice a day with some simple lotion, and instilling some weak atropine to promote healing. The patient must be warned of a possible recurrence.

A **foreign body embedded in the conjunctiva** is best removed by grasping the foreign body together with the piece of conjunctiva to which it is attached and cutting it off.

Burns of the cornea produce white areas of charred epithelial debris which are eventually thrown off; if not deep, they leave an abrasion, the treatment for which has just been described.

Caustic fluids of any kind produce much the same condition as is seen in a burn, a large abrasion resulting which may penetrate beneath

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the epithelium and into the superficial layers of the cornea. Some fluids give rise to greater damage than others; thus alkalis are more destructive than acids, the effects of the latter often leaving surprisingly little scarring.

Treatment.—I believe that the plan of treating injuries of this kind with neutralizing chemicals is merely theoretical, as the damage to the cornea is done almost immediately. We need not be deterred, however, from washing out the eyes with non-irritating lotions, for they give a considerable amount of comfort.

In all superficial injuries of the cornea, some oily solution such as liquid paraffin is beneficial, as it helps the lid to move easily over the globe and thus prevents the rubbing over the denuded area which causes so much pain.

In severe burns there is danger of adhesions forming between the lids and the globe (symblepharon), but this can be prevented by passing a glass rod daily between the palpebral and ocular conjunctiva to break down the smallest adhesion as soon as it forms.

2. **Penetrating wounds** of the eye are much more serious than the superficial ones, though less painful, and if the foreign body is retained the danger of subsequent complications is greatly increased.

When a **foreign body penetrates the cornea**, aqueous humour is evacuated. If the wound is small, only a little escapes, the lips of the incision soon coming together and remaining in apposition firmly enough to allow the anterior chamber to re-form. If the wound is large, the whole of the aqueous fluid is discharged and the iris is caught up and protrudes through the opening to a greater or less extent. It may be difficult to be sure that the iris is prolapsed, as the part that projects is usually covered with white lymph and loses its natural colour. An invariable sign is an oval-shaped pupil with the peaked end of the oval pointing towards the wound; with a little careful observation this cannot possibly be overlooked. The immediate **treatment** is to excise the prolapsed iris at the earliest possible moment, since the longer it is left the more difficult is it to deal with owing to the development of firm adhesions between it and the wound. After a week or ten days it is impossible to release it, and the risk of secondary complications is superadded. After gently separating the loose attachment to the lips of the wound with a repositor, the iris must be seized with iris-forceps close to the cornea and pulled out as far as possible, indeed much

farther than might appear necessary. While it is well on the stretch, cut it off close to the cornea, pressing slightly on the latter in the act of cutting. The cut ends will spring back into the anterior chamber free of the wound, which, provided that there is no infection, will heal up.

The foreign body may have **penetrated the lens**, in which case, if the wound in the capsule is large enough, aqueous humour will gain access to the lens matter in considerable quantity, and a traumatic cataract will result; this will be evident from the greyish-white reflex behind the iris and the loss of the red reflex from the fundus (see **EYE, EXAMINATION OF**). The lens matter will eventually become absorbed, or may be removed by curette evacuation.

When a traumatic cataract has developed there is an end to binocular vision, no matter how complete the final recovery may be; the wounding of the lens, therefore, is an important factor in the prognosis. Since it is impossible to determine for a few days whether the lens is injured or not, any opinion as to the future condition of the eye must be very guarded.

When a **foreign body is retained in the eye** it is desirable to remove it, but no attempt must on any account be made unless it can be seen or is capable of being attracted by an electro-magnet. It is advisable to have an X-ray photograph taken, to localize, if possible, its position in the globe. As both the necessary apparatus and the electro-magnet are usually impracticable for the practitioner, it will be best for him to send his patients to a large hospital in which such instruments are installed.

A piece of steel can be drawn forwards into the anterior chamber with the help of the magnet, assisted by a knowledge of its actual position as shown on the X-ray plate, and it can then be removed through a corneal section; but there is still some difference of opinion regarding the technique for removal if the magnet fails to extract the foreign body. Many foreign bodies, even steel, can be retained for years, sometimes permanently, without producing serious symptoms, so that if the vision is fairly good, and the eye quiets down well, it is far better left alone, for it is well known that a good many eyes are lost from meddlesome interference. Of the substances which penetrate the eye, glass is tolerated best and is the least dangerous; next comes steel, which can generally be removed; but

copper, wood, and stone lead generally to sacrifice of the eye, as they cannot be removed without damaging the structures connected with vision.

The **prognosis** in all the injuries described in this second group is uncertain for at any rate a few weeks, for, even if the foreign body is removed at once, there is always danger of the development of sympathetic inflammation, suppuration, and shrinking of the globe.

3. **Blows on the eye** from a large blunt instrument, e.g. a piece of wood or a fist, often produce much serious damage to its various structures. They may cause rupture of the globe, with or without a conjunctival wound, the former, of course, being more dangerous than the latter owing to the risk of infection. The rupture usually takes place at the thinnest part of the globe, just behind the sclero-corneal margin; the cornea itself very rarely gives way. There may be prolapse of the iris or dislocation of the lens into the interior of the eyeball or externally underneath the conjunctiva. The immediate result of such an injury is a large extravasation of blood into the eye, spreading all over the interior and hiding the iris from view, so that very few details can be made out in the early stages, and the red reflex of the fundus is lost (*see EYE, EXAMINATION OF*). Any prolapsed iris must be attended to at once, but as regards other structures it is well to desist from treatment until the blood has been absorbed; this happens comparatively soon, and the actual extent of the damage can then more easily be recognized. A dislocated lens is shown by the presence of a deeper anterior chamber than on the other side; it may be pushed back into the eyeball or extruded externally beneath the conjunctiva. No matter what its position, no attempt must be made to remove it, as such interference is always dangerous to the eye.

The immediate effects of such injuries are vitreous hæmorrhage, dislocation of the lens, traumatic mydriasis, laceration of the iris, irido-dialysis, and lowered tension, all of which are compatible with a degree of recovery with useful vision. Some of the remoter effects are traumatic cataract, irido-cyclitis, secondary glaucoma, suppuration of the eyeball (panophthalmitis), detachment of the retina, and rupture of the choroid.

Sympathetic irido-cyclitis is perhaps most to be feared, for it involves the possible loss of the presumably normal eye, and the judgment

required in deciding how far to temporise with the injured one is a matter of many years' experience. This subject is dealt with more fully in another place (*see UVEAL TRACT, AFFECTIONS OF*), but an eye that does not quiet down in about three weeks after an injury should be regarded with suspicion, even if there is still some sight remaining, and further advice must be sought before it is too late to save either eye. The question of excision always arises on these occasions, and the patient and his friends are often very persistent in pressing for an opinion on this point immediately after an injury. The practitioner must never commit himself as to whether an eye should be removed or not, nor be led into saying that it is in a hopeless state. Many eyes which in former years were not considered worth saving are now preserved as useful organs of vision.

MALCOLM L. HEPBURN.

EYELIDS, AFFECTIONS OF.—The affections here considered are (1) Blepharitis, (2) Sty, (3) Meibomian Cyst, (4) Blepharospasm, (5) Deformities, (6) Tumours.

1. **Blepharitis.**—Inflammation of the lid margin is a chronic septic condition which shows itself by redness of the edge of the lid. In the more severe cases the follicles of the eyelashes are infected and form ulcers, the lashes drop out or atrophy, the lid margin may become thickened and incurved—*entropion* (p. 477), and the lashes may rub on the cornea—*trichiasis* (p. 477).

Etiology.—The disease is common in children, especially of the poorer classes, and is essentially due to a staphylococcus; it is a frequent sequela of measles. It is maintained by dirty habits, e.g. rubbing the lids with unclean hands; in some cases the eyelashes are covered with nits, the ova of *Pediculus pubis*; in others an error of refraction, defective food assimilation, staphylococcal foci such as impetigo, septic tonsils, adenoids, or pediculosis capitis may be present.

Diagnosis.—The discharge from *conjunctivitis* may crust along the lashes and simulate blepharitis; the two conditions can be differentiated by cleaning the lid margin, which in conjunctivitis is normal. *Trachoma* must be excluded by evertting the lids and examining the palpebral conjunctiva; it may be present in the active granular stage, or in the passive scarred condition.

Treatment.—The lid margin must be kept clean by sufficient irrigation, followed by anti-

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septics in the form of an ointment. The lotions commonly used are lot. sod. bicarb., 10 gr. to the ounce, and lot. acid. bor. In chronic cases, zinc salts (zinc sulphate, $\frac{1}{2}$ gr. to the ounce of water) are useful, and sometimes the addition of three or four drops of liq. carbonis detergens to an ounce of lotion seems beneficial. Ung. hydrarg. ox. flav. dil., or ung. hydrarg. ammon. dil., should be well rubbed into the affected area by gentle massage. In bad cases the lashes should be cut short, and every few days the ulcerated margin may be rubbed with a pledget of wool on a glass rod, soaked in a 25-per-cent. solution of argyrol, 5-per-cent. protargol, or tincture of iodine.

Attention must of course be paid to general measures, and any error of refraction should be corrected by appropriate glasses.

2. **Stye** (*syn.* Hordeolum Externum).—An acute inflammatory infection of the sebaceous glands of an eyelash follicle. It commences as a small tender spot in the region of an eyelash. The neighbouring parts of the lid become œdematous, the skin over the hordeolum becomes hyperæmic, and a small abscess usually forms which discharges itself close to the line of lashes. In severe cases the inflammatory symptoms may be so extensive that œdema of the ocular conjunctiva (*chemosis*) is induced. Pain may be fairly acute. Some patients suffer from recurring hordeola, due to a diminished staphylococcal resistance, and it is well to examine for glycosuria.

Treatment.—Hot bathings every hour or every two hours should be carried out. For this purpose it is useful for the patient to bring the heat of nearly boiling water to the eyelid by means of a piece of lint or wool on the end of a wooden fork or spoon. This is dipped in the basin without the intervention of the fingers, and a greater heat can thus be applied. The treatment should be continued for some fifteen or twenty minutes at each application. Between the hot bathings a moist compress may be used with advantage. These measures generally suffice to cut short an attack, but in some cases an incision through the skin is necessary. In relapsing cases a staphylococcal vaccine is sometimes of value, and attention must be paid to the condition of the bowels and to the general health.

3. **Meibomian Cyst** (*syn.* Chalazion, Tarsal Cyst).—A chronic inflammatory swelling of a Meibomian gland, which breaks down into cystic material. The Meibomian glands are enormously enlarged sebaceous glands lying in

the substance of the tarsal plate; they open by small ducts whose orifices form a line along the lid border, between the *margo acuta* and the line of the eyelashes.

Signs.—A tarsal cyst forms a slowly progressive swelling in either the upper or the lower lid, and usually well away from the lid margin. It projects forwards so as to be visible as a smooth rounded swelling under the skin, to which it is not adherent except in late stages. On everting the lid it is seen that there is some swelling of the conjunctiva, which often has a bluish appearance where the underlying tarsus has been eroded.

Course and complications.—If left alone a tarsal cyst may gradually increase up to the size of a hazel-nut, but it more often suppurates earlier, forming an abscess of the lid (*Hordeolum Internum*), which ruptures through the conjunctiva and is followed by a suppurating granuloma. Apart from actual abscess-formation, a cyst may erode the conjunctiva, and granulation tissue may sprout through; there is then a good deal of muco-purulent discharge.

Diagnosis usually causes little difficulty. Cases with acute inflammation have to be diagnosed from other acute inflammations of the lids, such as *stye* or *acute dacryocystitis*. *Concretions in the canaliculus*, a rare condition, may also be mistaken for tarsal cyst, but if it is remembered that there are no tarsal glands on the nasal side of the punctum such a mistake will not arise.

Treatment.—Small inflamed cysts may occasionally resolve under hot fomentations, etc., otherwise operation is necessary. It consists of *incision and curetting*, and is generally somewhat painful, even after local anæsthesia; in nervous patients nitrous oxide may be necessary. If local anæsthesia is decided on, a few drops of 2-per-cent. cocaine are dropped on the conjunctiva, and then a little solid cocaine hydrochloride in crystal form is rubbed on the conjunctival surface of the swelling for about a minute. This usually suffices, especially if the cyst is large; but if it is small, it is well also to inject some 2-per-cent. solution of novocain under the skin about the tumour. The instruments required are Beer's triangular knife and a small sharp curette. After eversion of the lid, the conjunctival surface of the tumour is incised by the knife in a direction at right angles to the lid margin; the knife should be pointed away from the globe, and the cutting edge in the same direction (in case of sudden movement by the patient); the incision should

go well into the swelling, and the interior is then scraped out with the curette, so as to remove all granulation tissue from the wall of the cavity. A pad and bandage are applied for twenty-four hours, and boric-acid lotion may be used as necessary.

The patient should be instructed to use a boric-and-zinc lotion for some weeks afterwards, as there is little doubt that many of these cysts are due to a preceding conjunctivitis.

4. **Blepharospasm.**—Spasm of the orbicularis palpebrarum is most commonly a reflex due to some corneal condition, such as a phlyctenular ulcer. In other cases it is a purely functional disorder, and may be cured by a temporary *canthotomy*. In this operation one blade of a stout pair of straight scissors is passed behind the outer canthus, and the latter is divided by one snip. In blepharospasm due to corneal ulcer the main treatment is, of course, directed against the ulcer. In children the spasm is sometimes materially helped by holding the face and head under cold water forcibly for a minute or more.

5. **Deformities of the lids.**—(a) *Entropion*, or inversion of the lid margin, may be spastic (as with blepharospasm), senile, or cicatricial. If pronounced, it becomes associated with trichiasis, and then requires treatment.

(b) *Trichiasis*, or rubbing of the lashes on the cornea, is a further degree of entropion, or may be due to an error in the direction of the growth of lashes. It produces a vascular opacity and ulceration of the cornea, and therefore requires treatment. Its main causes are chronic blepharitis, conjunctival scarring from trachoma, lime burns, and wounds.

The treatment for entropion and trichiasis in simple spastic cases is by temporary sutures to keep the lid turned outwards. Senile cases require the removal of a horizontal strip of skin and muscle from the region close to the lashes. In scarred cases, epilation of the offending lashes by epilation forceps, destruction of the follicles by electrolysis, or some plastic operation is necessary.

(c) In *Distichiasis*, a congenital condition, the row of Meibomian ducts is replaced by an additional line of lashes.

(d) *Ectropion*, or eversion of the lid margin, may be due to chronic conjunctivitis, or scarring of the skin of the cheek or lids. It causes epiphora by eversion of the punctum, and chronic conjunctivitis by exposure. It may suffice to treat the conjunctivitis by appro-

priate measures, but often some plastic operation to the conjunctiva or cheek tissues is necessary. In cases where eversion of the punctum is the main feature, the epiphora may be relieved by dilating the punctum and slitting the canaliculus backwards for a distance of 2 mm.

(e) In *Lagophthalmos* the lower lid is so pulled down by a scar on the cheek that the lids cannot close, and an exposure ulcer on the lower part of the cornea is apt to occur. A plastic operation is usually necessary.

(f) In *Ankyloblepharon* the lids are partially united, while in *Symblepharon* the lid and globe are united owing to some preceding burn or injury. If it is extensive, a grafting operation will be required.

(g) *Ptosis* may be congenital or acquired, and in either case it may be partial or complete. The congenital cases are often bilateral, and associated with defective upward movement of the eyes; there is probably a mal-development of the muscle. In the unilateral cases there may have been a birth injury. If the pupil is covered an operation must be undertaken. Acquired ptosis is due to involvement of the third cranial nerve or of the cervical sympathetic (see OPHTHALMOPLÉGIA).

6. **Tumours of the lids.**—The more common tumours of the lids are rodent ulcer and dermoid cyst; these do not differ in characteristics from similar tumours elsewhere. Xanthelasma is considered separately. Primary Chancre, Epithelioma, Vaccinia Pustule, Nævus, Hæmangioma, Molluscum Contagiosum, and Plexiform Neuroma do not call for detailed description here.

F. A. JULER.

EYELIDS, EXAMINATION OF (see EYE, EXAMINATION OF).

EYESTRAIN.—Astenopia arising from the use of the eyes. It is dependent on a variety of causes, all of which may be aggravated by any condition leading to a lowering of the normal standard of health.

The cause of all eyestrain is fatigue and exhaustion of the muscular and nervous control of the mechanical adjustment of the sight. It may be due therefore to errors of refraction, muscle inco-ordination, e.g. latent squint (see STRABISMUS), errors in distribution of light, and faults connected with adaptation. It is not necessarily the quality or quantity of the light which is of chief importance, for symptoms of eyestrain may be produced by defects in the

FACIAL HEMIATROPHY

lids, pupils, etc., which affect the distribution of the light, and by variations in the amount and physiological properties of the retinal pigment and in fixation, etc., which influence adaptation.

The commonest cause is a slight departure from the normal refraction of the eye, which, although not sufficient to produce any inconvenience for distant vision, nevertheless demands increased and prolonged accommodative effort during reading or near work for a length of time. With slight errors of refraction, eyestrain is often met with also when the usual amount of accommodation is exercised at a time when the patient is in a weakened state of health. Thus it follows that small errors of refraction may never be noticed until circumstances arise which require constant application to near work, or until some severe illness has weakened the general muscular tone; this accounts for the complaint so often heard, that the eyes were perfectly well until after an illness.

Eyestrain also occurs if wrong glasses are ordered, or if glasses rightly prescribed are worn wrongly, for some degree of artificial astigmatism is in this way often added to the error already existing.

Symptoms.—Eyecache, headache in any part of the head, but most frequently over the forehead, worse after the use of the eyes and towards the end of the day, watering of the eyes, heaviness of the lids, desire to shut the eyes for a time after reading or sewing, are the symptoms most often complained of. To

errors of refraction are also ascribed many others which it is difficult to reconcile with any known defects in the ocular muscles, though the use of glasses apparently cures them. No greater mistake can be made than to ignore the symptoms complained of in these cases and regard them as trivial or of no importance.

The **diagnosis** is often very difficult, as the symptoms are entirely subjective in character and vary in different people; a thorough and careful examination is necessary to exclude each cause in turn.

Treatment.—The first step is to correct any refractive error which may exist; retinoscopy must be performed, generally under a mydriatic, and glasses prescribed first for reading, and later, if necessary, for constant wear. It is well known, however, that the amount of asthenopia bears no direct relationship to the degree of refractive error. If the muscular inco-ordination is found to be excessive, prisms must be ordered, in addition to glasses for the correction of the refractive errors. This requires considerable judgment, for no patient can tolerate prisms if they are beyond a certain strength, and operative measures may have to be considered.

Attention must also be paid to methods of illumination and to the amount of glare, since errors of distribution and faults of adaptation call for the use of extra-ocular muscles, e.g. the superciliary muscles, and this often leads to headache.

MALCOLM L. HEPBURN.

FACIAL HEMIATROPHY (*syn.* Parry-Romberg Disease).—A rare condition characterized by a progressive wasting of one side of the face. The wasting involves especially the skin, subcutaneous fat, and bones, the muscles being relatively less affected. It may attack the whole of one side of the face or be limited to some particular areas, as the orbital region and the cheek. Very rarely it is bilateral, or spreads to the neck or shoulder, or is associated with wasting in one side of the tongue and palate.

The disease, which usually begins in late childhood or early adult life, is seen in females more often than in males. It progresses slowly for a few years and then becomes

stationary. Its cause is unknown. In a few cases it has been attributed to injuries and infections about the face and throat, and to general diseases like scarlet fever and typhoid. Different theories have regarded it as due to a neuritis of the fifth nerve, as an affection of the cervical sympathetic fibres or ganglia, and as a congenital tendency showing itself in wasting at the situation of embryonal clefts. It causes no symptoms beyond the disfigurement. Its course is not influenced by treatment. In some cases the injection of paraffin or other substances has been resorted to in order to improve the facial appearance, but this procedure is not without risk.

P. W. SAUNDERS.

FACIAL PALSY

FACIAL PALSY (*syn.* Bell's Palsy).—Weakness of the movements of one side of the face may be due to a lesion of the upper motor neurones terminating in the facial nucleus, or to disease of the one facial nerve or its nucleus in the pons. The former type is usually part of a hemiplegia, the latter constitutes true facial palsy. This is most commonly due to a lesion of the nerve, which, as it has a long course through the temporal bone, is very liable to be damaged by septic diseases of the middle ear and by fractures of the skull that involve its petrous portion. The common "rheumatic palsy," which is generally attributed to cold or exposure, is a degenerative neuritis of the nerve in the Fallopian canal; by some it is regarded as an infectious condition. Syphilis, especially in its early stage, may involve the nerve here. Facial palsy is not infrequently associated with herpes in the trigeminal or cervical region; its cause is probably a swelling of the geniculate ganglion that compresses the nerve within the bony canal. Below the stylo-mastoid foramen paralysis may be produced by compression by enlarged glands or by tumours, and particularly by growths of the parotid gland. Lesions of the intracranial portion of the root are most frequently due to a gummatous meningitis. Within the pons it may be damaged by a tumour, hæmorrhage, or softening.

Congenital facial palsies also occur; they are probably due to a non-development of the nerve. It is occasionally injured at birth.

Symptoms.—Both sides of the face are rarely involved except in polyneuritis. In the ordinary rheumatic or otitic form the onset is usually sudden; it is frequently preceded or accompanied by pain in the ear, mastoid region, or cheek. All the muscles supplied by the nerve are generally paralysed; the folds disappear, the affected side becomes flat and expressionless, the mouth is drawn towards the sound side, and in longstanding cases the nose may be deviated to it too. The asymmetry becomes more obvious in movement; on showing the teeth it is only on the sound side that the lips are raised and separated; when the mouth is opened widely the arch of this upper lip appears flattened; the patient is unable to bring his lips firmly together; on attempting to blow out his cheeks, air escapes from the affected side, and in drinking, fluid dribbles from this corner of his mouth. Owing to palsy of the buccinator the palsied cheek flaps in and out with each respiration, and food

collects between it and the cheek. Articulation is disturbed, as the labials cannot be formed properly, especially at first when the muscles are toneless. The platysma does not contract on depressing the jaw. Paralysis of the orbicularis palpebrarum makes it impossible to close the eye, though the upper lid generally descends a little owing to relaxation of its levator; no winking occurs, and as dust and foreign bodies are not removed, conjunctivitis may result. On attempting to close the lids the eye rotates upwards and outwards (Bell's sign). The upper facial muscles are equally involved, and consequently the patient cannot wrinkle or corrugate this side of his forehead. Paralysis of the stapedius muscle may produce a hyperacusis, or greater sensitiveness to low-pitched sounds. Tear secretion is occasionally diminished, but the escape of tears on to the cheek is a greater inconvenience to the patient; it is due to the depression and eversion of the lower lid owing to lack of tone in the orbicularis. When the nerve is involved between the geniculate ganglion and the branching off of the chorda tympani, taste is lost on the anterior two-thirds of the same side of the tongue.

In incomplete palsies a feeble contraction of some or all of the muscles may be possible, or the paralysis may be local; only those supplied by its lower branch may suffer when the nerve is injured in the parotid region.

In the more serious cases, electrical examination towards the end of the second week reveals the reaction of degeneration in all the paralysed muscles; in slighter cases the reaction may be incomplete or almost normal.

As recovery takes place, contractures which exaggerate the natural folds may develop, so that while at rest the side of the face that is affected may be thought to be the normal, but the decision is always obvious on strong voluntary movement. Clonic or tic-like involuntary spasms are a rarer sequel of palsy. The deformity, as a rule, is less pronounced in cases of gradual onset.

Diagnosis.—Peripheral facial palsy is easily distinguished from an upper-motor-neurone weakness by the fact that in the latter the frontal and orbital muscles are little if at all affected, and the electrical reactions are normal. Hysterical palsy is very rare; the electrical reactions are unaffected, and it is usually only certain movements that are lost.

Treatment.—Treat the cause if possible. Surgical intervention is indicated when the

FÆCES, EXAMINATION OF

nerve is compressed by an accessible tumour, and some cases due to otitis improve when the ear condition is dealt with. Electricity is undoubtedly useful; moderate galvanic currents sufficient to excite contraction of the muscles should be employed from the end of the second week. Massage is also advisable. The overstretching of the paralysed muscles should be avoided, especially those of the cheek; this can be effected by a piece of stiff wire, one end of which, bent into a hook and covered by a bit of rubber tube, is inserted between the lips at the angle of the mouth, while the other end is passed round the root of the ear, like the limb of spectacles.

Grafting of the distal end of the facial nerve into the hypoglossal or spinal-accessory nerve has been tried in the absence of recovery; some good results have been obtained, but failures are more common. No effect can be expected for nine to twelve months at least.

GORDON HOLMES.

FACTITIOUS URTICARIA (*see* URTICARIA).

FÆCES, EXAMINATION OF. - - The **consistence** of fæces is chiefly determined by the amount of water they contain, but an abnormal softness may be due to an excess of fat, mucus, or swollen vegetable remains. An excess of fluid may arise from defective absorption or abnormal exudation, whilst delayed peristalsis with prolonged sojourn in the colon gives rise to hard fæcal masses or scybala.

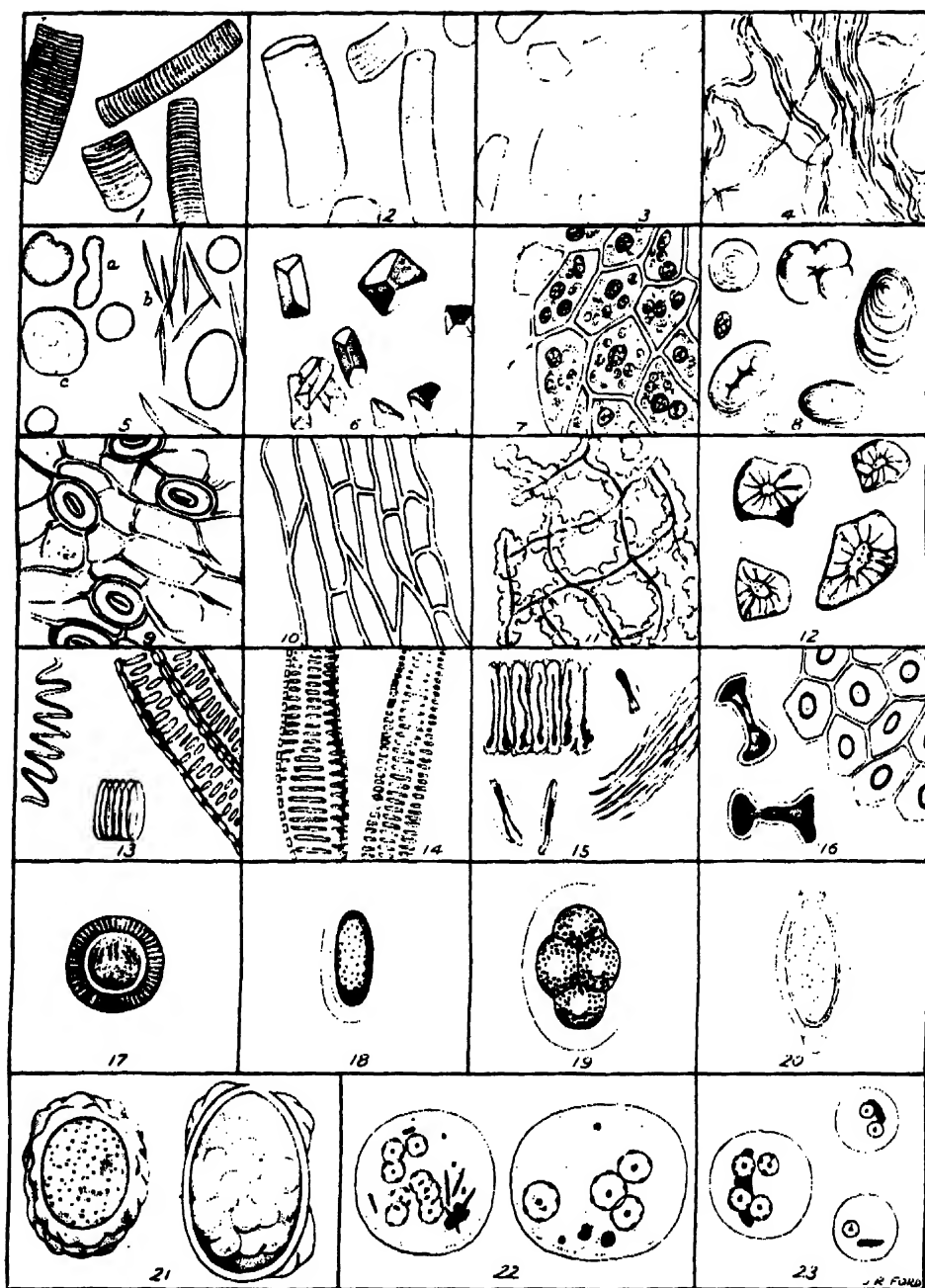
Shape and size.—The shape and size of solid motions is determined mainly by the size and tone of the anal orifice. Pencil- or ribbon-shaped fæces are produced by stenosis or muscular spasm of the rectum or anal orifice, or by paralysis of the lower part of the large intestine.

Colour.—The colour of the stools is influenced by the diet, the digestive secretions, discharges from the mucous membrane, and accidental ingredients. A vegetable diet generally produces fæces of a light colour, meat alone gives very dark-brown fæces, and milk gives yellow or yellowish-white stools. Certain fruits and vegetables, such as blackberries or spinach, may influence the colour.

Bile is the digestive secretion which chiefly affects the colour. In meconium and the stools of young infants, bilirubin is the normal pigment, but traces of biliverdin may appear, especially in the fæces of breast-fed children.

Distinctly green fæces in bottle-fed and breast-fed children are always pathological. In later life the bile pigments are normally reduced to hydrobilirubin (stercobilin), the presence of unchanged bilirubin or biliverdin indicating excessive peristalsis in the upper part of the intestine. The golden yellow colour of the stools in many diarrhoeas is due to bilirubin, but even normal-coloured fæces may be shown by chemical tests to contain unchanged bile pigment when there is catarrh of the upper intestine: thus assistance may be obtained in the diagnosis of duodenal ulcer. A small piece of the fresh fæces is thoroughly rubbed up with a concentrated watery solution of corrosive sublimate and left for twenty-four hours in a covered dish. If unchanged bile pigment be present, green particles are seen among the red or red-brown given by the hydrobilirubin. Obstruction to the flow of bile into the intestine gives rise to acholic or clay-coloured stools. This appearance is partly due to an absence or diminution of bile pigment, but the excess of fat, which is always present, contributes. A clay-coloured stool may contain a considerable amount of altered bile pigment when the obstruction is due to gallstones in the common bile-duct, but with cancer of the head of the pancreas even traces are rare. To demonstrate hydrobilirubin the corrosive-sublimate test may be used. A better method is to extract the fæces with ether to remove the fat, then with acid alcohol. The alcoholic extract is neutralized, mixed with an equal volume of a saturated solution of zinc acetate in alcohol, and filtered through a dry filter paper. The filtrate is then examined against a black background for the green fluorescence given by the pigment.

An **excess of fat** in the stools, causing them to be light-coloured or even white, may be due to other causes than lack of bile, such as advanced destructive lesions of the pancreas interfering with fat digestion, atrophy of the intestinal mucous membrane, and blocking of the lymph-channels by growths or tuberculous changes in the mesenteric glands, which prevents its absorption. Microscopic examination shows fat globules, fatty acid and soap crystals, but a quantitative estimation of the fat and a determination of the extent to which it has been digested are necessary before a reliable conclusion can be arrived at as to the presence of an abnormal amount and its probable cause. In pancreatic disease the undigested fat is generally in excess; whilst a



- 1, Muscle-fibre, undigested. 2, Muscle-fibre, partly digested. 3, Muscle-fibre residues.
 4, Connective tissue. 5, (a) Fat globules; (b) fatty acid crystals; (c) soap crystals.
 6, Triple phosphate crystals. 7, Rice starch in cellulose envelopes. 8, Undigested starch granules. 9, Epidermis of a leaf. 10, Cuticle of a seed. 11, Epidermis of plant stem.
 12, Woody cells of pear. 13, Spiral and pitted vessels of plant stem. 14, Scalariform vessels from woody tissue. 15, Palisade parenchyma from peas and beans. 16, Columnar cells of pea.
 17, Ovum of *Tœnia solium*. 18, Ovum of *Enterobius (Oxyuris) vermicularis*.
 19, Ovum of *Trichuris trichiura (Trichocephalus dispar)*. 20, Ovum of *Ancylostoma*.
 21, Ova of *Ascaris lumbricoides*. 22, Cysts of *Entamoeba coli*. 23, Cysts of *Entamoeba histolytica*.

PLATE 12.—SUBSTANCES FOUND IN FÆCES.

FÆCES, EXAMINATION OF

Relative excess of combined fatty acids (soap) is suggestive of defective absorption. Clay-coloured stools without jaundice may occasionally arise from the reducing action of bacteria on the hydrobilirubin, which converts it into a colourless compound.

The most important exudate from the intestine affecting the colour of the stools is **blood**. When it comes from the rectum or sigmoid flexure, blood usually retains its normal colour; but when from the large intestine, the hæmoglobin is changed into hæmatin and, if present in sufficient amount, gives the fæces a black or even a tarry appearance. Small quantities intimately mixed with the fæces are referred to as "occult blood," and are most important in diagnosis. They do not affect the appearance of the stool, and can only be detected by chemical means. The *benzidine* test is the best. A small portion of the fæces is ground up with water to form a thin emulsion and boiled to destroy ferments. While it is cooling, a knife-point of pure benzidine (Merck) is dissolved in 1 or 2 c.c. of glacial acetic acid. From 3 to 10 drops of the emulsion are added and the mixture shaken. Hydrogen peroxide (3 per cent.) is then cautiously added, the emulsion being shaken after each addition, until a blue or green colour develops, or some 2 c.c. of the reagent have been used. As the test is exceedingly sensitive, giving a distinct colour with as little as 1 in 250,000, it is advisable that the patient should have been on a hæmoglobin-free diet for several days before the test is made, and accidental sources of blood, such as bleeding from the gums or from piles, must be excluded. A positive result under these conditions on four or five consecutive days is suggestive of malignant disease of the gastro-intestinal tract, bile-ducts, or pancreas; while the intermittent presence of occult blood points to the existence of a simple ulcer.

Mucus in the fæces, in macroscopic masses, generally comes from the colon; small particles, only recognized by microscopical examination, usually come from the small intestine. In the latter case the mucus is generally bile-stained, and the deeper the pigmentation the higher in the bowel is its probable source.

Pus rarely gives a distinct yellow, or grey, tint to the stools unless it comes from the lower part of the large intestine.

Some **drugs** modify the colour of the fæces. Bismuth causes black or dark grey-green stools; iron only changes the colour after the stool has been exposed to the air, when it becomes

blackish-grey; calomel may cause greenish stools from the presence of unreduced bile-pigments; rhubarb, senna, and santalin cause yellow stools; while kino leads to bright-red, and hæmatoxylin to violet-red, fæces.

Reaction.—Normally the fæces are faintly alkaline or amphoteric in reaction. Any condition which increases putrefaction causes them to become more alkaline, whereas intestinal fermentation and imperfect digestion of carbohydrates and fats render them acid. The fæces in advanced pancreatic disease and acute enteritis, for instance, are often strongly acid, while the stools in colitis are strongly alkaline.

Microscopical examination of a thin emulsion of the fæces shows normally only vegetable tissue, crowds of bacteria, a few fatty acid and soap crystals, amorphous debris, sometimes a few triple phosphate crystals and, occasionally, one or two partly digested muscle-fibres. The presence of connective tissue is usually pathological, and points to imperfect gastric digestion (Plate 12). Numerous well-preserved muscle-fibres are most commonly met with in disease of the pancreas, especially carcinoma, but may also be due to other causes. Numerous fat globules suggest disease of the pancreas. Fatty acid and soap crystals in excess indicate defective digestion and absorption of fats. Starch granules are rare and point to catarrh of the intestine. Epithelial cells, erythrocytes, and pus cells are not recognizable, as a rule, unless they come from the lower part of the bowel, when their presence indicates inflammatory changes and ulceration. The discovery of the ova of intestinal worms is one of the most important uses of the microscope in fæcal examinations. Those most commonly met with are *Enterobius (Oxyuris) vermicularis*, *Trichuris trichiura* (*Tricocephalus dispar*), *Tania solium*, *Tania mediocanellata*, and *Ancylostoma duodenale*. Amœbæ are most readily to be found in particles of mucus, especially those which are bloodstained. These should be picked out of the fresh, warm fæces and mixed with normal saline on a slide. A cover-glass, supported at the side by a large hair, is applied, vaselin is painted round the edges, and the preparation is examined with an eighth- or sixth-inch objective. The slide should be kept at about the body-temperature by a warm stage or by standing the microscope on a steam radiator. The addition of 1 per cent. watery methylene-blue stains epithelial cells blue, but does not stain the amœbæ. P. J. CAMMIDGE.

FAVUS

FÆCES, IMPACTION OF (*see* CONSTIPATION).

FAINTING (*see* SYNCOPE).

FAIRLEY'S REACTION (*see* SEROLOGICAL DIAGNOSIS).

FALLOPIAN TUBES, DISEASES OF (*see* GONORRHOEA; HYDROSALPINX; SALPINGO-OÖPHORITIS).

FAMILY PARALYSIS, PERIODIC (*see* PERIODIC PARALYSIS, FAMILY).

FARADISM (*see* ELECTRICAL TREATMENT).

FAT-EMBOLISM (*see* LUNG, EMBOLISM OF).

FATTY HEART (*see* MYOCARDIAL DEGENERATION, PROGRESSIVE).

FATTY LIVER (*see* LIVER ENLARGEMENT, DIFFERENTIAL DIAGNOSIS OF).

FAVUS.—A disease of the skin, hair, nails, or mucous membranes, caused by a parasitic fungus.

Etiology and pathology.—Favus is rare in England, and comparatively common in Scotland. In both countries the cases are diminishing in number. The disease occurs almost exclusively in alien immigrants, principally those from Russia and Poland. School children are most liable to become infected, and boys more than girls, but the disease may begin at any time of life and may persist to old age. The degree of infectivity is low, and one member of a family may suffer without infecting the others. Insanitary conditions and ill-health are predisposing factors. The two chief varieties of fungi causing human favus are (1) *Achorion schönleini*, to which the great majority of cases of ordinary favus of the scalp and skin are due, and (2) *A. quinckeanum*, derived from the mouse, which does not attack the scalp but causes a few cases of favus of the glabrous skin. Possibly cats, dogs, and birds may be the medium of contagion in exceptional instances. The fungus, which may be examined under the microscope in liquor potassæ or after staining with aniline gentian-violet and Gram's iodine, consists of a mass of wavy branching mycelium and round or oval spores, the hair-shafts being filled with long mycelial tubes, a few spores, and often some air-bubbles. The parasite invades the epidermis at the mouth of a hair-follicle and proliferates around the hair (PLATE 28, Fig. 4, Vol. III, facing p. 138), which is attacked

secondarily. Eventually the pressure exerted by the growth of the fungus causes complete atrophy of the hair. Cultures show a yellowish ridged surface, those of *A. quinckeanum* being white and downy with concentric rings.

Symptomatology.—The common situation for favus is the scalp. It begins insidiously as a minute yellow point or as a scaly spot sometimes resembling a vesicle or a pustule. In a typical case a pea-sized sulphur-coloured disc develops around a hair and becomes depressed in the centre, forming a cup-shaped crust or "scutulum." On the glabrous skin favus gives rise to circinate scaly or sometimes vesicular patches, upon which scutula may be seen. The patches may spread extensively over the body, and in rare cases invade the mucous membranes. Mouse favus causes similar ringworm-like lesions with or without scutula. Favus of the nail begins with yellow spots or streaks at the side or free edge, and gradually destroys its substance. With the exception of slight itching or discomfort, there are no subjective symptoms.

Diagnosis.—The yellow concave scutula and the peculiar stale mousy odour they emit when present in abundance are characteristic features. When scutula are not formed or have been removed by treatment, the presence of grey lustreless hairs which do not break off as in ringworm but can be pulled out entire, showing a swollen root-sheath, and of cicatricial bald patches, taken in conjunction with the long history of the disease, will enable the diagnosis to be made; it will be confirmed by the finding of the fungus in the hairs or crusts or by the growth of cultures. These features will enable the disease to be distinguished from other scaly conditions such as eczema and psoriasis, from diseases causing bald patches like alopecia areata, pseudo-pelade, and lupus erythematosus, and from syphilitic or traumatic scarring. When the nails are affected, cultures and the presence of mycelium in the scrapings must be the deciding features.

Prognosis and treatment.—Favus of the scalp is extremely intractable, and shows no tendency to heal spontaneously. Total epilation by X-rays in conjunction with the application of antiseptic lotions and ointments is much better than epilation with forceps, and will save many months or years of trouble. Favus of the glabrous skin presents no great difficulties, but when it affects the nails avulsion is the best treatment. S. E. DORE

FILARIASIS

FEEBLE-MINDEDNESS (see IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY).

FEEDING OF INFANTS (see INFANT FEEDING).

FEIGNED DISEASES (see MALINGERING).

FEIGNED ERUPTIONS (see ECZEMA).

FERMENTS (see PATHOLOGY, CHEMICAL, MODERN DEVELOPMENTS OF).

FIBRINOUS BRONCHITIS (see BRONCHITIS).

FIBRINOUS PNEUMONIA (see Lobar Pneumonia, under PNEUMONIA).

FIBROID DEGENERATION OF THE HEART (see MYOCARDIAL DEGENERATION, PROGRESSIVE).

FIBROID POLYPI (see UTERUS, POLYPI OF).

FIBROIDS, UTERINE (see UTERUS, NEW GROWTHS OF).

FIBROMATA OF THE SKIN (see SKIN, FIBROMATA OF).

FIBROSIS OF THE HEART (see MYOCARDIAL DEGENERATION, PROGRESSIVE).

FIBROSIS OF THE LUNG (see LUNG, FIBROSIS OF).

FIBROSITIS (see MYALGIA).

FIFTH NERVE (see TRIGEMINAL NERVE).

FILARIASIS.—Invasion of the body by one or other species of worms of the genus *Filaria*, a group of nematode helminths.

Etiology.—Though invasion by any filaria should, strictly speaking, be included under this head, of late years the tendency—at least as regards the human subject—has been to limit the term filariasis to those filariæ whose young or embryonic forms circulate in the peripheral blood. Of these, four are known in man, namely *Filaria bancrofti*, *F. loa* (PLATE 24, Figs. 6, 5, Vol. II, facing p. 487), *F. perstans*, and *F. demarquayi*. The last two do not give rise to pathological symptoms, so they need not be further discussed. *F. loa* (*Loa loa*) may also be dismissed in a word. Its adult forms live in the subcutaneous and connective tissues, and in their wanderings about may cross the eye. In such a case they are seen wriggling under the conjunctiva, and in many instances have been removed from this site; while here they cause lachry-

mation, smarting sensations, and minor degrees of conjunctivitis. In other instances the passage of the worm under the skin gives rise to fugitive swellings popularly known as Calabar swellings. These are about the size of a pigeon's egg, appear suddenly, are not associated with inflammatory changes, and disappear again of their own accord in a few days. The distribution of the worm is a very restricted one, its geographical area being confined to the West Coast of Africa in the vicinity of the Equator.

To *F. bancrofti* we owe a train of pathological maladies, some of great gravity. The adult worms of this species inhabit the lymphatic system—the lymphatic glands or lymphatic channels. They measure some inches in length, and, being about the thickness of thin catgut, are easily visible to the naked eye. The males and females breed, the latter ultimately giving birth to vast numbers of young embryos which pass along the lymph-channels and are poured into the blood. These forms remain here, and do not undergo any further development until sucked out of the blood by suitable mosquitoes. When this occurs, a development or metamorphosis takes place in the thoracic muscles of the insect, on the completion of which the enlarged embryos pass into the proboscis and, when the mosquito bites, back into the tissues of man. From the subcutaneous tissues they pass into the lymphatics, develop further, become sexually mature, and start their cycle again. The embryos circulating in the peripheral blood are harmless; the trouble comes entirely from the adults in the lymphatics. The geographical distribution of this species is very widespread throughout the tropics.

Pathology.—As the parasites inhabit the lymphatic glands and vessels, the lesions which they cause necessarily pertain to the lymphatic system. In some instances, as in so many other helminthological infections, little or no damage appears to be done by a small or even fairly large invasion of the worms. For example, if one examines the night-blood of one hundred apparently healthy persons in a filarial district with no signs of filarial disease about them, fifteen or twenty of these may show embryos, an evidence that adult filariæ are present somewhere in the body. Though there may be no signs of the disease at the time of the examination, in many instances, if the cases are followed up, pathological lesions ultimately develop. It is questionable

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if the parasite is really ever harmless, but symptoms depend upon where it is lying: if in some unimportant lymphatic, the damage done there may not be appreciable; if in an important one, grave changes are produced. The adult worm acts pathologically in four ways:

1. A collection of the parasites, coiled up together, may plug a lymphatic and produce thrombosis and consequent blocking of the channel.

2. It may cause inflammatory changes in the vessel with subsequent fibrosis and thickening of the walls, this eventually leading to a great narrowing, or even complete occlusion, of the lumen.

3. If it inhabits the lymphatic glands, inflammatory changes are set up there; evidence of this being seen first in cellular infiltrations and secondly in the production of dense fibrous tissue which ultimately completely shuts off the passage of lymph through the glands.

4. Death or destruction of the parasites may form a nidus for septic organisms—staphylococci or streptococci—with consequent abscess-formation or general sepsis.

Cases have been reported in which the thoracic duct itself was blocked, a general varicosity of the lymphatic system below the obstruction resulting. The adult worms usually select, however, the lymphatics which drain the legs and genitals, and those in the pelvis and lower abdomen or in the epididymis, and cause lesions in these parts.

The arms and upper parts of the body are much more rarely attacked. Blocking of the lymphatics and glandular system, with repeated attacks of inflammation superadded, ultimately leads to chronic hypertrophic changes known by the name of elephantiasis.

Symptomatology.—The following list of diseases shows how diverse are the clinical manifestations of filariasis:

1. Filarial lymphangitis.
2. Dilated lymphatics, superficial or deep.
3. Chyluria and lymphuria.
4. Genital troubles—
 - (a) Lymph scrotum.
 - (b) Orchitis.
 - (c) Epididymitis.
 - (d) Hydrocele.
 - (e) Chylocele.
5. Coccidial invasions—
 - (a) Filarial abscess.
 - (b) Purulent lymphangitis and adenitis.
 - (c) Septicæmia.
6. Elephantiasis.

1. **Filarial lymphangitis.**—This condition is a very frequent one in filarial districts, and may be the first sign of infection. The most common sites for its occurrence are the legs and spermatic cord. A patient, previously healthy, is seized suddenly with rigor, vomiting, and often delirium, attended by great pain in the groin and femoral region and swelling and inflammation of the leg. An examination reveals a condition in many ways resembling erysipelas. In a white person the affected limb, generally the foot and leg below the knee, is hot, red, and swollen, and inflamed lymphatics are seen running up to the groin, while the glands in the femoral region are enlarged and very tender to pressure. After lasting for three to four days, all the symptoms gradually subside, the leg returns to its normal shape and size, and the attack is over. People who have once suffered are, however, very liable to subsequent attacks, and after many of these some permanent thickening of the affected part appears, which is often the starting-point of elephantiasis (*see below*).

When the spermatic cord is affected the general symptoms—rigor, vomiting, and delirium—are very severe, and the pain, which radiates up into the abdomen and down into the testicles and legs, is excruciating.

2. **Dilated lymphatics.**—After, or even without, an attack of lymphangitis, small dilations may sometimes be seen on some of the superficial lymphatics of the limbs. Some of these cyst-like swellings have been excised and living adult filariae found in them. Lymphatic dilations are more commonly detected in the groin, where they may take on a very large size, simulating hernia in some respects. These large, baggy, fluctuating masses are only part of a general varicose condition of the lymphatics of the pelvis, their production being due to the blockage of main lymphatic trunks high up in the abdomen. They contain chyle or lymph, and sometimes embryonic filariae.

3. **Chyluria and lymphuria.**—This is a part of the general condition of dilated lymphatics mentioned above. The lymphatics supplying the kidneys and bladder are dilated, and some rupture, pouring their contents into the urine. If the obstruction is in the thoracic duct, chyle will be present; whereas if lower down in the lymphatics, before they join the receptaculum, only lymph. The chief clinical sign of the condition is the milky appearance of the urine; if blood is also present the colour will

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be reddish-white. The condition intermits. An attack may last for a short or a long time (months) and then disappear, but a relapse is sure to follow sooner or later. It is specially frequent in filariated women after parturition. Associated with the passage of the abnormal constituents there are pain and aching in the back, and sometimes retention of urine due to clots. If the milky urine is allowed to stand in a urine glass, the lymph coagulates and finally three layers may be determined—a creamy layer at the top, a white coagulation in the centre, and a reddish deposit at the bottom. Embryo filariæ may or may not be present. Chyluria may occur alone or be associated with other filarial manifestations such as dilated lymphatics, or, if the patient is a male, with some of the genital symptoms now to be described.

4. Genital troubles. (a) *Lymph scrotum*.—In this condition the scrotum is enlarged and small dilated lymphatics may be seen on its surface, many of these exuding lymph or chyle. It feels soft to the touch. Later on, when inflammation has been superadded, it may become hard and elephantiasis may set in. (b) *Orchitis and epididymitis*.—As already mentioned, a favourite site for the adult filariæ is the epididymis. When here, the parasites readily set up inflammatory troubles and, if the testicles are attacked, orchitis as well. The lesions resemble in their symptoms ordinary gonorrhœal epididymitis. (c) *Hydrocele* in filarial countries is especially common and often owes its etiological basis to the filariæ. (d) *Chylocoele* is a condition where chyle or lymph is present instead of clear serum in the tunica vaginalis.

5. Coccid invasions.—If a filaria dies in some superficial lymphatic and organisms settle down at the spot, an abscess results. After being opened such collections of pus quickly heal up and give little trouble. On the other hand, when they occur in the abdomen serious results follow. In some instances, fortunately rare, a purulent focus arises around a worm lying in some abdominal lymphatic, the supuration spreading backwards along the dilated lymphatics till ultimately these become full of pus. The patient then dies of septicæmia. Pus appearing in a gland in the femoral region or groin will give rise to a bubo. In evacuating such collections of pus, fragments of the adult worms may be found, and in countries where filariasis exists they should always be looked for.

6. Elephantiasis.—The usual sequence of events in the development of this condition is as follows: The patient suffers from repeated attacks of filarial lymphangitis. After each of these a little thickening is left, and gradually increases until the condition becomes definitely one of elephantiasis. Any part of the body may be attacked—the legs, penis, scrotum, labia majora, arms, breast, and skin of the scalp, while pedunculated masses may even grow out from the groins. The affected part is hard and solid, the skin, especially in natives exposed to the weather, becoming rough and verrucose, whilst ulcers are apt to develop in the sulci about the ankles. The scrotum may attain an enormous size, some of the very large ones almost touching the ground as the patient stands. Attacks of lymphangitis in the affected parts are frequent.

Diagnosis.—The diagnosis in some of the forms of filariasis is not always easy, though in others it is plain sailing. Sometimes the finding of embryos in the peripheral blood helps, but, on the other hand, their absence means little, as after the lesions have developed either the adults may be dead, or the obstruction set up prevents the embryos from getting into the peripheral blood. After attacks of lymphangitis it is unusual to find embryos, and this is almost always the case in elephantiasis. In the other forms they are found more commonly. The possibility of filariasis should always be considered in hydroceles and testicular troubles in people coming from abroad.

Prognosis.—Where definite lesions have developed the prognosis is not a good one, the tendency being for these to get progressively worse. This is especially so in elephantiasis. In other cases the condition may remain stationary for considerable periods of time.

Treatment.—This is not satisfactory. No drug is known which will kill the adult worms *in situ*, and when obstructive lesions have arisen in the lymphatics little or nothing can be done to relieve them. Antimony tartrate (tartar emetic) injections intravenously have recently been tried, but so far the results are not hopeful. If acute lymphangitis be present, the patient should be confined to bed and may have some mild antiseptic dressing applied to the limb. For chyluria, again, rest in bed is the main line of treatment. Where pus forms and is accessible it should

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be removed on ordinary surgical lines. Epididymitis and orchitis may be greatly relieved by morphia. Large dilated lymphatics in the groin are much better left alone. For elephantiasis surgical procedures have been tried. These are especially successful in scrotal cases, the large masses seen there being capable of complete removal with good results. Cutting out strips of skin and subcutaneous tissues and other procedures have been adopted for elephantiasis of the limbs, but none of these measures does very much good. If the condition is very advanced, amputation may be required. (J. C. Low.

FINSEN LIGHT (see LIGHT TREATMENT).

FISTULA, FÆCAL (see PERITONITIS, TUBERCULOUS).

FISTULA FROM MALFORMATIONS OF UMBILICUS (see UMBILICUS, MALFORMATIONS OF).

FISTULA IN ANO (see ANAL FISTULA).

FISTULA IN GENITAL PASSAGES (see URETHRAL STRICTURE; VAGINAL AND UTERINE FISTULÆ).

FITS (see CONVULSIONS; EPILEPSY).

FLAT-FOOT (see TALIPES).

FLATULENCE. — Flatulence may occur simultaneously in the stomach and the intestine, but in many cases it is confined to the stomach and less frequently to some part of the intestines. In each case it may be due to (a) excessive production of gas by fermentation or putrefaction, (b) the introduction of excess of air by aerophagy, and (c) deficient elimination.

GASTRIC FLATULENCE

Etiology.—(a) *Excessive fermentation* in the stomach is never sufficiently active to give rise to flatulence unless pyloric obstruction is present. Deficient hydrochloric acid does not itself result in increased bacterial activity in the stomach; thus, flatulence is not a symptom of achylia gastrica, in which the rate of evacuation is generally increased.

(b) *Aerophagy* is the commonest cause of flatulence. It is generally associated with nervous dyspepsia, but it also occurs with organic diseases such as gastric and duodenal ulcer and gall-stones. The patient feels some

slight discomfort in the stomach, which he thinks is due to "wind," and which he imagines he can "disperse" by eructation; as there is really no excess of gas present, the attempt proves unsuccessful, but results in the swallowing of air. After half a dozen or more attempts have been made without success, air being swallowed on each occasion, the stomach becomes so distended with air that an attempt is at last successful. The excessive salivation which often occurs in gastric disorders associated with hypersecretion and in septic conditions of the mouth also leads to flatulence, as air is swallowed with each mouthful of saliva. The severest cases of aerophagy occur independently of dyspepsia in women who are always intensely neurotic, the symptom being purely hysterical.

(c) *Deficient elimination of gas* occurs when absorption is diminished owing to the obstruction of the portal circulation which occurs in cirrhosis of the liver and heart failure; swallowed air and the gases produced by fermentation are insufficiently absorbed, and severe flatulence results.

Symptoms.—The most common symptom resulting from gastric flatulence is a sensation of fullness in the upper part of the abdomen, especially under the left costal margin. The abnormal accumulation of gas in the stomach pushes up the diaphragm; this may cause palpitation and pseudo-anginal attacks. Dyspnoea results in those who are predisposed by such conditions as asthma and cardiac weakness, and flatulence may also be the immediate cause of an attack of true angina pectoris.

Diagnosis.—When a patient complains of "flatulence" it is first necessary to ascertain whether excess of gas is really present. This can be done most readily and accurately by means of the X-rays, as it is often difficult to distinguish by percussion whether an accumulation of gas is in the stomach or the splenic flexure of the colon.

Pseudo-flatulence may be due in neurotic individuals to spasm of the diaphragm: a sensation of great distension is suddenly experienced, and the abdomen becomes so enlarged that the clothes have to be loosened. The distension disappears as suddenly as it came, without eructation or passage of flatus; it also disappears under an anæsthetic and on firmly compressing the whole of the abdomen for some minutes. The absence of excess of gas in the stomach can at once be detected

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with the X-rays, which also show the low position of the diaphragm and the absence of respiratory movements. The condition can be cured by teaching the patient to breathe properly with her diaphragm. Pseudo-flatulence is more often due to the patient misinterpreting the sensation of fullness which is caused by the increased intragastric pressure produced by hypertonus or the presence of excess of solid or liquid contents, as well as by excess of gas. There is no abdominal distension nor increase in gastric resonance, and the patient is unable to bring up any gas, but the condition is often complicated by aerophagy.

The gas brought up in cases of aerophagy is odourless and tasteless, but it may be offensive when it is produced by fermentation. Aerophagy is probably present if eructation is frequently repeated; the diagnosis is certain if it occurs six or more times in rapid succession, as fermentation cannot give rise to such a large quantity of gas. Eructation occurring before breakfast in the absence of pyloric obstruction is always due to aerophagy, as there is nothing in the stomach from which gas could be produced. The diagnosis can be confirmed by means of the X-rays, with which it is easy to watch the whole process of aerophagy.

Treatment.—The treatment of flatulence due to excessive fermentation is that of the pyloric obstruction. When flatulence is due to aerophagy, it is often only necessary to explain to the patient the cause of his trouble in order to cure him. He should be told not to eructate, however much he may desire to do so. If he finds it very difficult to restrain himself, he should open his mouth or clench his teeth upon a cork whenever the desire is very strong, as it is then difficult to swallow air, though any excess of gas in the stomach can be expelled. When aerophagy is due to dyspepsia, this requires appropriate treatment. Momentary relief, sufficient to help the patient to forgo eructation, can generally be obtained by sipping chloroform water, but the use of spirits for this purpose should be strictly prohibited.

In severe cases of hysterical aerophagy isolation is required. If simple explanation and persuasion fail, a stomach-tube should be passed and kept in the stomach for some minutes, the patient having previously been told that the treatment will give her permanent relief.

INTESTINAL FLATULENCE

Etiology.—(a) *Excessive production of gas* in the intestines is due to excessive fermentation or putrefaction, but the amount of gas produced by the former is much greater than that produced by the latter. Excessive fermentation and putrefaction occur when, for any reason, the normal digestion of carbohydrates and proteins respectively is deficient, so that abnormally large quantities of one or the other reach the colon, where they undergo bacterial decomposition. Infection of the intestines with pathogenic organisms, which cause either fermentation or putrefaction as a result of their activity, is an additional cause which can act even when digestion is otherwise normal.

(b) If aerophagy continues when the stomach is empty, some of the air passes into the small intestine, in which the greater part is absorbed, but in severe cases a little may reach the colon and be finally expelled from the rectum as almost odourless flatus.

(c) Absorption from the intestines as well as the stomach is greatly diminished when the venous circulation is obstructed in cirrhosis of the liver and in heart failure. It ceases entirely when the venous circulation is completely arrested in strangulation of a part of the intestine.

In constipation a part of the colon may contract so firmly upon the faeces it contains that the gas which is behind it cannot pass; retention of gas then occurs in addition to retention of faeces.

Symptoms.—Intestinal flatulence gives rise to borborygmi and to a sensation of fullness and pressure in the abdomen. As gas accumulates most commonly in the splenic flexure, where the distension may cause palpitation and dyspnoea by pushing the diaphragm upwards, the discomfort is often mistaken by the patient for gastric flatulence, and he consequently makes an effort to relieve himself by eructation; as there is no excess of gas in the stomach, his efforts result in aerophagy. Distension of the intestines acts as a stimulant to their contractions, so that flatulence is often associated with colic, which is at once relieved by passing flatus.

Diagnosis.—The only symptom which proves conclusively that a sense of fullness in the abdomen is due to intestinal flatulence is its disappearance as a result of passing an excessive quantity of flatus. When a patient with symptoms pointing to the presence of

FŒTUS, MEASUREMENTS OF

excess of gas under the left dome of the diaphragm finds that the passage of flatus gives him relief, but eructation does not, it is probable that the cause is a collection of gas in the splenic flexure; by means of the X-rays it is easy to ascertain whether excess of gas is present in this situation. In order to determine whether intestinal flatulence is due to excessive fermentation, excessive putrefaction, or some other cause, the stools must be examined in the way described under **DIARRHŒA**.

Treatment.—The treatment of intestinal flatulence consists in removing the cause.

A. F. HURST.

FLOATING OR MOVABLE KIDNEY
(see **VISCEROPTOSIS**).

FLOODING (see **MENORRHAGIA** AND **METORRHAGIA**, CAUSES OF).

FŒTAL DEFORMITIES (see **MONSTERS**).

FŒTAL ŒDEMA (see **ŒDEMA**).

FŒTUS, MEASUREMENTS OF.—The principal measurements of the **average full-term fœtus** are:

Length, 20 in. (50 cm.)
Weight, $7\frac{1}{2}$ lb. (3½ kilos).
Length of cord, 20 in. (50 cm.).
Bisacromial diameter, $4\frac{3}{4}$ in. (12 cm.).
Bitrochanteric diameter, 4 in. (10 cm.).

The *diameters of the head* are:

Occipito-frontal, $4\frac{1}{2}$ in. (11.5 cm.).
Suboccipito-frontal, 4 in. (10 cm.).
Mento-vertical, $5\frac{1}{2}$ in. (13.5 cm.).
Submento-vertical, $4\frac{1}{2}$ in. (11.5 cm.).
Submento-bregmatic, $3\frac{3}{4}$ in. (9.5 cm.).
Suboccipito-bregmatic, $3\frac{3}{4}$ in. (9.5 cm.).
Biparietal, $3\frac{3}{4}$ in. (9.5 cm.).
Bitemporal, $3\frac{1}{4}$ in. (8 cm.).
Bimastoid, $2\frac{3}{4}$ in. (6.5 cm.).

The length and weight of the fœtus at **different stages of development** are (Galabin and Blacker):

Month	Length	Weight
1st	$\frac{1}{2}$ in. (1 cm.).	
2nd	$1\frac{1}{2}$ in. (3 cm.).	240 gr. (15.4 grm.).
3rd	$3\frac{1}{2}$ in. (8 cm.).	3 oz. (84.9 grm.).
4th	$5\frac{1}{2}$ in. (14 cm.).	$7\frac{1}{2}$ oz. (204 grm.).
5th	9 in. (22.5 cm.).	1 lb. (450 grm.).
6th	12 in. (30 cm.).	$2-2\frac{1}{2}$ lb. (900-1,100 grm.).
7th	15 in. (37.5 cm.).	$3-4$ lb. ($1\frac{1}{2}$ kilos).
8th	17 in. (42.5 cm.).	5 lb. (2½ kilos).

FOLIE CIRCULAIRE

Haase's rule for determining the relation between the age and the length of the fœtus is as follows:—

- 1 During the first five four-week months, the square of the month gives the length in centimetres.
- 2 During the second five four-week months, the age in months multiplied by 5 gives the length in centimetres.

A. W. BOURNE.

FOLIE CIRCULAIRE AND RECURRENT INSANITY.—A mental disease of which the outstanding features are the periodicity of the symptoms and their widely opposed character at different parts of the cycle. It usually consists of three stages—one of excitement, one of depression, and one of comparative sanity. These usually occur in the same order in each attack, and the cycle may continue for many years. In other cases the excitement and depression follow each other immediately, without any sane interval.

In recurrent mania or melancholia the patient suffers, at irregular intervals, from a succession of attacks similar in character and in symptoms.

It has been questioned whether pure mania and melancholia exist—whether all these cases should not be classed under manic-depressive insanity; but though in most cases there tends to be a swing of the pendulum in the opposite direction after the acute attack, cases are found in which maniacal attacks recur again and again without being followed by states of depression; and the same may be observed in periodic melancholia. Typical cases of folie circulaire or of manic-depressive insanity are not so common as recurrent mania and melancholia.

Etiology.—As in other mental diseases, a neuropathic heredity renders the subject more vulnerable and liable to react to causes which would not affect ordinary persons. Folie circulaire is more frequently found in the intellectually cultured classes than among manual workers. Exhausting illness, prolonged worry, fatigue and mental strain, fear or starvation, may act by producing malnutrition and exhaustion of the nervous system. Arteriosclerosis is often found associated with it, and may be a cause, or both conditions may be the result of some toxin. It is more frequent in men than in women, and the first attack occurs most often between the twentieth and thirty-fifth years, but it may start later in life. Some-

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times, though very rarely, attacks of mania or melancholia are seen in young children.

A tendency to periodicity is noticeable in many physiological and pathological processes, and many sane individuals exhibit throughout life distinct periodic changes in mood (*cyclothymia*) but without ever passing beyond the limits of self-control. In circular insanity these variations are exaggerated to a marked degree, and, during the extremes of an attack, asylum treatment usually becomes necessary.

Symptomatology.—The onset may be in the form either of mania or of melancholia; sometimes a brief period of depression precedes a long maniacal attack.

Maniacal stage.—Often the first sign of exaltation is an abnormal activity. Patients cannot rest, and must always be up and doing. The quantity of work done may be great, but the quality is apt to be indifferent; powers of concentration are diminished and nothing is done thoroughly. The patient becomes extravagant and morbidly generous, and shows a lack of reticence. One who is normally quiet, reserved, and modest becomes loquacious, often witty and acute, showing quick reasoning powers and a readiness to talk about himself and his affairs to all and sundry. As the excitement increases there is a tendency to eccentricity in dress, "dressing up" and decorating; there may be sexual excitement, and there is always emotional instability, the patient laughing and sometimes crying on slight provocation, and the temper becoming irritable and violent. There is boundless self-confidence: patients will boast of talents and accomplishments which they do not possess; those who have the most limited powers of music may volunteer to display them and be incapable of realizing their failure. The motor restlessness finds expression in rubbing the face, dancing, jumping, etc. In the subacute stage it is sometimes usefully got rid of by hard work in house or garden, or by walking. Usually sleep is short and broken, and a large amount of food is taken. Patients become untidy and careless in dress, and often coarse in language, and those who in their normal state are refined and reticent may talk openly of sexual matters and masturbate without shame or concealment.

Melancholic stage.—In the slightest form there may be only a simple psychic inhibition, without hallucinations or delusions. The patient is mildly depressed, finds it difficult

to think, cannot collect his thoughts or follow the train of ideas in a book or in conversation. When the attack is more serious he becomes absolutely self-centred and loses interest in his surroundings; everything appears in an unfavourable light, he distrusts himself, and has a feeling of unworthiness, often an overwhelming sense of sin. The slightest failings in the past are magnified into crimes, and a marked feature is the blunting or absence of affection for his family or friends. Patients lose all sense of comfort or help from religion, and feelings connected with it are coloured by sentiments of fear, hopelessness, and remorse for imaginary crimes.

The slightest exertion costs an effort; the patient feels unable to work, shrinks from meeting other people, and has no sense of humour. At last he gives up all activity, sits silent, sometimes moaning or wringing his hands or repeating that "everything is wrong," "there is no hope," or that he "cannot live." There is a feeling of fear, horror, or intense mental pain; sleep is broken, food becomes distasteful and is often entirely refused, and the idea of suicide presents itself as the only refuge from misery. This danger should constantly be remembered when treating melancholia.

Among the commonest delusions are those of being altered into an animal or devil, and organic ones, such as that the patient has no stomach, that the bowels are stopped up, that he cannot swallow, etc. The feelings of discomfort caused by the constipation which is always present may suggest these ideas, and a careful physical examination is essential in order that any cause may be found and attended to.

In some cases there is undoubted alimentary auto-intoxication, and the mental recovery is coincident with improved digestion and excretion. In others, some painful disease, such as cancer, may be the starting-point of organic delusions.

Auditory hallucinations are uncommon, but some melancholics hear "voices" which urge them to commit suicide, and during an acute attack of mania transient visual or auditory hallucinations may occur.

Diagnosis.—The diagnosis of a case of folie circulaire from an attack of simple mania or melancholia can be made only after the whole series of phases has been observed. Any attack of acute excitement is liable to be followed by some depression due to physical

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exhaustion, but this alone is not an indication that the case is a circular one. If, however, after the depression has been recovered from, the patient shows signs of excitement again followed by depression, it becomes probable that the case is assuming a circular form. Similarly, if recovery from an attack of mania or of melancholia is followed in a comparatively short space of time by a relapse, there is a probability that the case is of the recurrent type.

From dementia præcox.—Successive attacks of excitement in the course of dementia præcox may resemble recurrent mania, but usually the patient does not make such a good recovery between attacks, and close observation may show signs of catatonia, or other characteristic symptoms. The personal and family history are helpful.

From general paralysis.—Attacks of excitement or depression occur in general paralysis, but a diagnosis will be made from the physical signs and a positive Wassermann reaction in the cerebro-spinal fluid.

Prognosis.—The prognosis of circular or recurrent insanity is grave as regards mental recovery. A patient may suffer from mania or from melancholia, recover, and never have another attack, but where a definitely alternating condition is observed, or the attacks recur at regular intervals, complete recovery is rare, though there may be long remissions. Persistent delusions and hallucinations are an unfavourable sign. Violence and excitement tend to become less as years go on, and the extremes are less marked. When there is definite arterio-sclerosis, or senile changes appear, a weak-minded condition may supervene, by which the original disease is masked; but as a rule there is little tendency to dementia, even when the attacks continue for many years.

Treatment.—Treatment must be symptomatic, and directed towards building up physical health and the resistive power of the nervous system. The more the sane interval can be prolonged, the better the chance of resisting the onset of another cycle. During the maniacal stage quiet surroundings are most valuable, and often isolation from friends or other patients allays the excitement. Asylum treatment is almost invariably indicated during the extremes of an attack, especially during the depressed stage, when the danger of suicide is always present. Each case must be treated on its merits: in some

FOREIGN BODIES IN AIR-PASSAGES

the stores of nervous and muscular energy must be worked off by hard physical exercise; in others exhaustion develops early, and requires as much rest in bed as possible,—at least ten hours out of the twenty-four should be spent in bed and as much time as possible in the open air. Plenty of nourishing food, especially such as is fattening, is essential. Alcohol and much meat should be avoided. Should food be refused, tube feeding should be resorted to without hesitation, and, when given in this way, large doses of olive or cod-liver oil (as much as 1 to 3 oz. t.d.s. of the latter) can be tolerated, and act beneficially both in fattening and in relieving the constipation.

When sleeplessness is troublesome, a continuous warm bath (20–30 minutes at a temperature of 98° F. just before bedtime) should be tried. Sometimes an adequate dose of calomel calms excitement better than ordinary sedatives. Maniacs usually have a low blood-pressure and are poorly nourished, so tonics are indicated, such as iron, arsenic, nuxvomica, and quinine. During the depressed stage, cheerful surroundings, varied occupation, and the presence of other people are valuable. Constipation is often very serious, and should be treated vigorously. Paraldehyde is a useful hypnotic; sulphonal and trional are effective, but should not be used for more than three or four days at a time. Adalin is valuable and safe. In melancholia, especially in the agitated form, opium sometimes acts like a charm, allaying restlessness and soothing mental pain, but, needless to say, the patient should not be aware of what drug he is taking. The liquor meconicus (Squire) is one of the most satisfactory forms and should be given in doses of 20–30 min. together with tinct. hyoscyami 30 min., three times a day; the taste is easily disguised with tincture of orange.

E. M. JOHNSTONE.

FOLLICULIS (*see* TUBERCULIDES).

FOOD POISONING (*see* POISONS AND POISONING).

FOREIGN BODIES IN THE AIR-PASSAGES.—Foreign bodies may be present in the nose, the naso-pharynx, the pharynx or larynx, the trachea or bronchi.

1. IN THE NOSE.—This condition is usually met with in children, the commonest substances being pellets of paper, peas, beans, beads, and fruit stones.

FOREIGN BODIES IN THE AIR-PASSAGES

Symptomatology.—In the early stages there is slight irritation or pain, later on unilateral irritating discharge, leading to excoriation of the *alae nasi* and upper lip. On examination the mucosa is found congested and swollen, usually completely hiding the foreign body. The nose is full of muco-pus, occasionally bloodstained, or there may be a thick membranous exudation looking like diphtheritic membrane.

Diagnosis.—In children the condition must be diagnosed from *fibrinous rhinitis*; as a rule the use of a probe will detect a foreign body. In adults it must be distinguished from various types of *ulceration*, simple or malignant. Here again, after the application of a little cocaine and adrenalin, a probe will establish the diagnosis.

Treatment.—Cleanse the nostril with a spray and apply cocaine and adrenalin, which will reduce the congestion and render the parts anæsthetic; then pass a strabismus hook up into the middle meatus and bring it down behind the body, which, as a rule, can gently and firmly be extracted by this means. In children it is wiser to give a general anæsthetic to avoid violent struggling.

2. IN THE NASO-PHARYNX.—Foreign bodies get into this position but rarely, and then usually as the result of vomiting. They can easily be seen by posterior rhinoscopy, and can be removed by postnasal forceps.

3. IN THE PHARYNX AND LARYNX. The bodies met with fall into two categories—the large, usually a tooth-plate or bolus of food; and the small, such as fish-bones, pins, etc.

Symptomatology. In the case of large bodies the symptoms are usually urgent, being those of asphyxia. With small bodies in the pharynx the usual complaints are of pain and irritation. If in the larynx there is in addition a paroxysmal cough, and occasionally glottic spasm with signs of asphyxia.

Diagnosis.—Usually a clear history is given by the patient of a sharp pain during deglutition, to which are added the above symptoms. A careful and detailed investigation must be undertaken. The tonsils, base of the tongue, valleculæ, pharynx, and larynx must each be examined thoroughly. If some time has elapsed since the body was impacted, it will probably be buried in swollen mucous membrane. Any such swollen area requires to be painted with adrenalin and cocaine to reduce

the swelling, and may require palpation with the finger in order to detect the foreign substance. Frequently the offending body, after causing a tear in the mucous membrane, passes down and is swallowed; the symptoms, however, generally persist for some days. In the case of metallic substances an X-ray examination may afford valuable aid.

Treatment.—In cases of large impacted bodies, with urgent symptoms of asphyxia, immediate treatment is imperative. If the patient is unconscious and has stopped breathing, laryngotomy or tracheotomy must at once be performed, and artificial respiration resorted to. If, however, he is still breathing, though showing signs of asphyxia, the mouth should be opened and a cork inserted between the teeth; it may then be possible with a finger to remove the body. If this is not possible a tracheotomy must be performed and the removal done under general anæsthesia. A bolus of food can either be hooked out with the finger, or broken up and pushed down the œsophagus. The smaller bodies can, as a rule, be removed quite easily with straight or bent forceps, with or without cocaine, according to the position. In the larynx the majority can easily be extracted, under cocaine, with laryngeal forceps. If, however, it is found that the body cannot be so removed—as when it is impacted across the glottis, or in one of the ventricles—a thyrotomy becomes necessary.

When no foreign body can be detected after careful examination, the patient should be ordered a bromide mixture and lozenges of menthol and cocaine.

4. IN THE TRACHEA AND BRONCHI.—If the foreign body has traversed the glottis it almost always passes into a bronchus—usually the right. In seven cases which the writer has had, the substances met with were a glass bead, a pin, a shawl-pin $2\frac{1}{2}$ in. long, a cherry-stone, all in the right bronchus, and a boot button, a tin-tack, and a piece of rubber tubing which had slipped off a gag, in the left.

Symptomatology.—A cough is always present, and differs from that of a foreign body in the larynx by being constant and not intermittent and paroxysmal. Dyspnoea usually occurs. There may be hæmoptysis if the foreign body is sharp or pointed. Pain is occasional.

On auscultation respiratory signs may be wanting in the portion of lung supplied by the

FOREIGN BODIES IN THE ALIMENTARY CANAL

bronchus; it is well to remember that smooth, round bodies, such as beads, occlude the bronchus more completely than irregularly shaped bodies. Occasionally a dry r le may be heard at the point where the foreign body is impacted.

The **prognosis** is bad unless the body is soon removed. Bronchitis, pneumonia, abscess-formation, gangrene, and bronchiectasis are some of the complications which may follow its inspiration. Moreover, unless it is extracted within a few hours the mucous membrane swells up and renders removal much more difficult.

Treatment consists in removal by either upper or lower bronchoscopy. (*See also RESPIRATORY PASSAGES, ENDOSCOPIC EXAMINATION OF.*)

J. GAY FRENCH.

FOREIGN BODIES IN THE ALIMENTARY CANAL.—Swallowed foreign bodies lodge most often in the pharynx,  sophagus, or rectum. In general, it may be said that a foreign body which reaches the stomach has a fair chance of passing through the whole alimentary tract. This is true even of sharp or angular bodies. The immunity of the alimentary canal from damage by irregular objects is surprising. The diagnosis of the site of impaction is important. In the  sophagus some pain or difficulty in swallowing, or the sensation of something in the throat, may only be indicative of an abrasion of the mucous membrane. A reliable history in the case of children, and even quite frequently in adults, is wanting. The first step, therefore, if the swallowed object is opaque to X-rays, is to submit the patient to a screen examination.

When the foreign body is seen in the  sophagus, an attempt, under general an sthesia, should be made to extract it with the  sophagoscope as soon as possible. This is by far the best procedure in skilled hands, though it must always be regarded as fraught with danger should the  sophagus be softened by inflammation from the foreign body having remained impacted for three or four days. But in such cases the passage of any instrument is a risky undertaking. Only when  sophagoscopy is unavailable from lack of the instrument or a skilled operator should the older methods be employed. The "coin-catcher" is very successful in dislodging coins; it is passed carefully beyond the coin and as carefully and slowly withdrawn. The "ex-

panding probang," which is passed beyond the object before being opened, and then withdrawn, may be used for irregular bodies and pins, which are caught in the bristles.  sophagotomy is indicated only when other methods have failed. It is a serious operation with a high mortality, and is nearly always followed by a protracted convalescence.

When the foreign body has passed into the *abdomen* there is no urgency to take steps for its removal, spiked and irregular though it may be. The writer has watched an open safety-pin pass through the alimentary canal of a child of two without doing any damage. The patient should be kept under frequent observation, all aperients forbidden, and a diet giving much residue (brown bread, porridge) prescribed. Liquids must be limited in amount. Cotton-wool torn up into small pieces and swallowed has been recommended. By these means the angularities of the foreign body become encased in indigestible material, and the mucous membrane is thereby protected. The indications for operative removal are: (1) Cessation of the onward passage of the foreign body, as shown by the X-rays; (2) signs of intestinal obstruction; (3) signs of commencing perforation of the wall of a viscus. The last two are indications for operation without delay. From the stomach or intestine the foreign body is removed by gastrotonomy or enterotomy.

When the foreign object lies in the *rectum*, where it can be felt by digital exploration and where it may cause severe tenesmus and pain on def cation, it should be removed through the anus as soon as possible. Under full an sthesia the anus is dilated; a small Sims vaginal speculum is then inserted by which the coccyx is drawn backwards. The forward curve of the coccyx is the real obstacle. The coccyx being pulled strongly backwards, the foreign body (which may be quite large when it is introduced per anum) is grasped with forceps and removed. Should the coccyx have lost its mobility, its resection should be the first step of the operation. This is done through a longitudinal incision over the dorsum of the coccyx. The bone is exposed, the sides and the tip freed from muscular and ligamentous attachments, the anterior surface cleared, and removal completed by bone forceps. No harm results from its removal. Only rarely have the anus and rectum to be split in the median line posteriorly to provide sufficient room.

C. A. PANNETT.

FOREIGN BODIES IN THE EAR

FOREIGN BODIES IN THE EAR.—The removal of a plug of *impacted cerumen*, which may first be considered, is best effected by syringing the ear with lukewarm water after the cerumen has been softened by a solution of hydrogen peroxide (10 vols. per cent.), a few drops of which may be instilled into the ear twice a day for two or three days.

Foreign bodies proper are usually met with in children. Immediate removal is unnecessary, except of bodies like peas or beans, which are liable to swell and cause pain, and those causing coughing or vomiting. Most of the serious consequences are due to unskilful efforts at extraction. The commonest foreign body is cotton-wool, which is easily and safely removed with a pair of aural forceps.

The practitioner's first concern should be to ascertain the nature of the body. If, for example, it is a lead pencil, or some such object, with the point inwards, syringing the ear, which is the usual treatment, is inadvisable, as it tends to propel the pencil farther into the meatus and to lead to impaction.

Syringing should be performed with an ear syringe, to the nozzle of which an inch or two of fine rubber tubing is attached. The canal should be lubricated with oil, and lukewarm water used, preferably sterilized. In the case of peas or beans which have become swollen, the previous instillation of alcohol drops to reduce their bulk is advisable.

For foreign bodies which are impacted, Politzer suggests the following plan: The walls of the canal and the end of the foreign body are carefully dried. A camel-hair brush charged with a concentrated solution of glue is brought into contact with the foreign body. On the glue drying, the foreign body adheres to the brush, and can be pulled out.

Should these efforts prove unavailing, the case should be handed over to the expert. *On no account* (save in the case of cotton-wool) *should forceps be employed*. Smooth spherical bodies especially, such as beads or peas, easily escape from between the blades of the forceps, as they are being closed, into the depths of the bony meatus, and even through the membrane into the middle ear, and acute suppuration is set up with all its dangers.

The safest instrument to use, if expert aid is not available, is a gently curved blunt ear-spud. Under bright illumination—the patient, if a child, being anaesthetized—the spud is cautiously passed along the postero-superior meatal wall until it comes to lie between the

FOREIGN BODIES IN THE URETHRA

tympanic membrane and the foreign body. The spud is now pressed downwards and forwards to engage the foreign body, and then drawn slowly outwards and the foreign body with it.

In some instances, especially when rude efforts at extraction have been made, painful otitis externa results, with narrowing of the meatus by swelling or granulations, a condition for which surgical measures are necessary.

Insects in the ear may be floated out by filling the meatus with oil. DAN M'KENZIE.

FOREIGN BODIES IN THE EYE (see EYE, INJURIES TO).

FOREIGN BODIES IN THE URETHRA.

—These include a calculus descending from the bladder, an object (slate-pencil, match, pin) introduced from without by a child or a lunatic, or the end of a catheter which has broken off. Micturition is always interfered with, and may be accompanied by bladder tenesmus. Sometimes there is complete retention. The foreign body can be felt from the outside when it is lodged in the anterior urethra. In the case of a smooth, cylindrical object, attempts at removal from the meatus may be made. Fix the foreign body by compressing the urethra against the pubic arch behind the impacted object, otherwise it may be pushed by the manipulations into the bladder—an occurrence to be avoided. Then pass urethral or sinus forceps down to the foreign body, seize it, and with the utmost gentleness withdraw it. This manoeuvre is to be done under visual guidance through a urethroscope if possible. Another method is to take a catheter with a lumen greater than the diameter of the foreign body and, having cut off its end, to insert it down to the object, which, with the aid of the fingers externally, it is induced to ensheath. The catheter and object can then be withdrawn together, external manipulation assisting. When the object is very irregular, or has been impacted some time, removal through the meatus leads to too much laceration of the mucous membrane. In such cases an incision is made on the under surface of the penis, the foreign body removed, and the urethra and overlying tissues sutured with catgut, except the skin, for which silkworm gut is used. A pin in the urethra usually lies with the point directed towards the meatus. Under anaesthesia the point is made to penetrate to the skin on the

FOURTH DISEASE

under surface of the penis. It is then seized, drawn out until the obstruction due to the head is felt, and redirected so that the head now lies towards the meatus, when it can be grasped with forceps and withdrawn. A hair-pin is removed in a similar fashion, both extremities being made to perforate together and the loop being turned towards the meatus.

A foreign body impacted in the prostatic urethra can often be pushed on into the bladder, and then removed by suprapubic cystotomy, or, if it is a stone, crushed by the lithotrite. When it cannot be dislodged from the prostatic urethra, median perineal section must be done, care being taken to incise the urethra behind the compressor urethræ muscle. The urethra should not be sewn up after this operation.

C. A. PANNETT.

FOREIGN BODIES IN THE VAGINA (see VAGINITIS).

FOURTH DISEASE.—This was a provisional name suggested by Dr. Clement Dukes, of Rugby, for an acute exanthem which he regarded as distinct from scarlet fever, measles, and German measles. Its existence is almost universally discredited by physicians of large experience of the eruptive fevers, and the following description, which is taken from Dukes' original paper (*Lancet*, 1900, ii, 89), would apply either to the scarlatiniiform variety of rubella or to mild cases of scarlet fever:—The season of prevalence is mainly the spring. The incubation period may range from nine to twenty-one days. As a rule, there are no prodromal symptoms, but in sharp attacks there may be slight sore throat and malaise before the rash appears. The first symptom usually noticed is the rash, which is scarlatiniiform, and covers the body in a few hours. Desquamation usually follows, but bears no relation to the intensity of the eruption. The throat is swollen and the conjunctivæ are pink. All the lymphatic glands are enlarged, hard, and tender, the posterior cervical, axillary, and inguinal being chiefly affected. The temperature ranges from 98.4° to 103° or 104° F., and the pulse is affected accordingly. Nephritis is rare, and there are practically no sequelæ. Recovery is the rule. The duration of infectivity is about a fortnight.

J. D. ROLLESTON.

FRACTURES FROM THE MEDICO-LEGAL STANDPOINT (see INJURIES FROM THE MEDICO-LEGAL STANDPOINT).

FRIEDREICH'S DISEASE

FRAGILITAS OSSIUM (see OSTEOGENESIS IMPERFECTA).

FRAMBÆSIA (see YAWS).

FRECKLES (see LENTIGO).

FREQUENCY OF MICTURITION (see URINE, VARIATIONS IN AMOUNT OF).

FRIEDREICH'S DISEASE (*syn.* Hereditary Ataxy).—This disease, which has a tendency to affect more than one member of a family but is rarely hereditary, usually commences in childhood or adolescence, and has a slowly progressive course. It is characterized by inco-ordination of voluntary movement with little or no loss of muscular power till its late stages, abolition of the tendon-jerks and extensor plantar responses, nystagmus, disturbance of articulation, and skeletal deformities. The pathological changes to which these disturbances are due are degenerations of the dorsal columns of the cord and of the spinocerebellar and pyramidal tracts. It is generally assumed that these degenerations are dependent on an inherent defect in the constitution of the fibres. Males are more commonly affected than females.

Symptomatology.—The onset of the disease is always slow and gradual. The first symptoms commonly appear between the ages of 6 and 10 years, but are sometimes present in early childhood or may be delayed till puberty or later. The first sign is usually an awkwardness in gait and a tendency to fall and stumble; walking becomes slowly more irregular and unsteady and acquires a drunken character. The difficulty in maintaining balance is most obvious in turning and on rising quickly from a seat. As the disease advances, the legs become feeble, and finally the patient, if he survive long enough, is bedridden. There is also a difficulty in maintaining fixed attitudes; in standing, the patient sways from side to side, and there are usually tremors of the head and trunk. Romberg's sign is frequently present.

The arms also become ataxic, but usually considerably later than the legs; it is their finer actions that are chiefly affected, but in severe cases the inco-ordination may be so great that the patient is unable to use his limbs. Strength is seriously reduced only in the later stages of the disease. The tone of the muscles is generally diminished, but the legs may be spastic. Contractures are rare.

The knee-jerks and other tendon reflexes are abolished—their absence may be the first sign

FROSTBITE

of the disease—and the plantar responses are invariably extensor. The sphincters generally escape. There are seldom pains or other subjective sensory disturbances. The state of sensation varies; as a rule there is some blunting of cutaneous sensibility on the legs, and the sense of position and the appreciation of movement are reduced here. Nystagmus is not so frequent or pronounced a symptom as it is assumed to be, but it is often present in advanced cases; the ocular movements are otherwise normal. Speech is often slurred and scanning, and occasionally explosive. The most common and characteristic skeletal deformities are lateral curvature of the spine, generally associated with some kyphosis, and talipes equino-varus. Intelligence is rarely impaired, but some patients become dull and childish. Congenital heart disease, especially pulmonary stenosis, is common in Friedreich's disease, and is occasionally responsible for sudden death.

Treatment.—We know of nothing that arrests the disease, but tonic treatment and attention to the health of the patient enable him to keep about longer than would otherwise be the case. Re-educational exercises on Frenkel's system often improve the gait.

GORDON HOLMES.

FRÖHLICH'S SYNDROME (see PITUITARY GLAND, AFFECTIONS OF).

FRONTAL SINUS, DISEASES OF (see SINUSES, ACCESSORY AIR, DISEASES OF).

FROSTBITE is due to the withdrawal of more heat from the tissues than they can lose without disturbance of their function. The degree of cold and the duration of exposure necessary to produce frostbite vary greatly. The robust and vigorous withstand much more exposure than the aged and feeble, or the young with a poor circulation. The extremities are most affected.

Four degrees of frostbite, analogous to the four degrees of burns, are recognized. In the first degree hyperæmia alone results, in the second degree vesicles develop, in the third degree sloughing of the tissue takes place, and in the fourth degree total death of the limb or part of the limb occurs.

In the *first* degree there is pain, followed by loss of sensation in the affected part, which becomes pale. When recovery takes place, œdema and a bluish-violet coloration appear, which last for a few days. An unsightly red-

FURUNCULOSIS

dening of the tips of the nose or ears may remain permanently. Chilblain (q.v.) or *pernio* is a variety of frostbite of this degree. In the *second* degree, in thawing, the cooled part becomes dark red or purple. Œdema comes on and blebs appear. The blebs dry up, form crusts, and no scarring occurs unless infection takes place. In the *third* degree the tissues are killed. Not much reaction is seen when thawing takes place, though vesicles may appear. The dead area remains a deep blue, and in it there is neither circulation nor sensation. Dry gangrene follows, a line of demarcation develops, and the slough is shed. The surrounding tissues are affected to the first and second degree. From the healing of the ulcer scarring and contractures occur. In the *fourth* degree the frostbitten part is frozen hard, and is so brittle that a nose or a finger may be broken off like a piece of glass.

Treatment.—Warmth should very gradually be restored to the part. Sudden warming is very painful and also very dangerous; stasis and thrombosis are likely to occur and produce gangrene, which might have been avoided. It is best to rub the part with snow and then with cold water, which may be gradually warmed. An hour or two may be consumed in the process. As the temperature returns the pain and burning sensation do so also. The venous congestion must be relieved by elevation of the part. When thawing has taken place, the injured area must be made surgically clean and treated exactly like a burn (see BURNS AND SCALDS). Pain which prevents sleep may require morphine, and is lessened if the affected part is not allowed to get too warm. The sinusoidal current and diathermy have been extolled by some as curative agents.

C. A. PANNETT.

FUNCTIONAL MURMURS (see HEART, FUNCTIONAL MURMURS OF).

FURRED TONGUE (see STOMATITIS AND GLOSSITIS).

FURUNCULOSIS.—An acute circumscribed staphylococcal infection about one or more hair-follicles, manifesting itself either as a boil or as a carbuncle.

Etiology and pathology.—In most cases the causal organism is the *Staphylococcus pyogenes aureus*, but in rare cases *S. pyogenes albus*. The condition may begin as an impetigo, infection subsequently taking place along a hair-follicle. Anæmia, renal disease, debility,

GALL-STONES

constipation, and diabetes predispose to furunculosis.

Symptomatology.—In the earliest stages a *boil* consists of a hard, tender, circumscribed mass. Later pus appears and discharges, leaving a central necrotic core. The axillæ, forearms, glutei, and beard region are especially liable to attack; on the nape of the neck, a rather common site, the condition is often associated with comedones, and here local inoculation by collars, etc., may prolong the disease. Boils tend to appear in crops separated by intervals of longer or shorter duration. They are not in themselves dangerous except on the face, and especially the upper lip, when secondary cerebral infection may occur.

A *carbuncle* is a deep spreading infection of the skin due to a staphylococcus. As the result of local necrosis, multiple openings occur through which pus is discharged. A large slough forms, which separates, leaving an ulcer. Carbuncles are most often found on the neck, shoulders, and buttocks; the condition is a serious one.

Diagnosis.—Boils must be distinguished from inflammatory *ringworm of the beard*. They are often associated with *scabies*. *Anthrax* and *actinomycosis* should be kept in mind.

Treatment.—Diabetes, albuminuria, and constipation, especially the latter, must, if present, be treated. Fresh yeast, a teaspoonful thrice daily, often acts well in furunculosis; it should not be given when vaccines are employed. A mixture of dilute sulphuric acid with sulphate of magnesia is often efficacious.

Sublimed sulphur in drachm or half-drachm doses, or fresh calcium sulphide in small doses, may be prescribed as an alternative to sulphuric acid. The affected area should frequently be washed with weak perchloride or boric lotion to prevent auto-inoculation. In the early stages boric fomentations are necessary. A boil may often be aborted by the application of a mercury-and-carbolic plaster in which a small central hole has been made. An incision is only indicated when pus has definitely formed, and it should be a small one. Squeezing is always undesirable.

Vaccines are employed in all stages of boils. The initial dose should be 100 millions (*Staphylococcus aureus*), gradually increased to 250 millions at intervals of a week. To prevent relapse it is advisable to continue vaccine treatment for some weeks after apparent cure. Stock vaccines are usually satisfactory, but in rebellious cases autogenous ones are required. Two other useful remedies are collosol manganese and stannoxyl. The former is given by injection, the latter in tablet form. Collosol manganese seems to have a special action in bringing about the retrogression of the furuncle.

A *carbuncle* may be freely laid open under anaesthesia and scraped. The general condition should be improved, and vaccines administered as in boils. Injections of collosol manganese are often extremely beneficial.

H. MACCORMAC.

FUSED KIDNEYS (see KIDNEY, CONGENITAL ANOMALIES OF).

GALL - BLADDER, INFLAMMATION OF (see CHOLECYSTITIS).

GALL-STONES (Cholelithiasis).—The presence of gall-stones in the gall-bladder or bile-ducts.

Etiology.—There are two groups of factors at work in the production of gall-stones: (1) direct local causes, (2) predisposing causes.

(1) **Direct local causes.**—The immediate cause of the formation of gall-stones is an infection of the biliary system. Stagnation or thickening of the bile alone will not lead to precipitation of cholesterol nor of bilirubin-calcium/

Inflammation of the bile-ducts produces an albuminous exudate which precipitates bilirubin-calcium, and accounts for the presence of the calculi formed entirely of this substance which are found in the intrahepatic ducts.

Most of the stones formed in the gall-bladder are composed largely or wholly of cholesterol, which is excreted in excess by the cells of the mucous membrane lining it, when they are in a condition of catarrhal inflammation. Some cholesterol is present in the circulating blood combined with an oleic radicle and is excreted into the gall-bladder as cholesteryl oleate. The oleic radicle is split off and absorbed, leaving

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the cholesterin in the bile. The cholesterin formed locally, however, is much more important than that excreted into the gall-bladder.

The infection which leads to gall-stone formation is usually of low virulence, and the chief bacteria concerned in this catarrhal inflammation are the *Bacillus coli*, *B. typhosus*, and the paratyphoid group; these organisms have been found many times in the nuclei of recent and even of old calculi. *B. coli* reaches the gall-bladder by the portal vein and not by an ascending inflammation of the common bile-duct. In typhoid and paratyphoid fevers there is always a generalized hæmic infection, and, although their organisms may be conveyed in some cases from the intestinal lesions by the portal vein, the infection generally occurs by means of the hepatic artery, and is derived from the systemic circulation.

(2) **Predisposing causes.**—Anything which interferes with the emptying of the gall-bladder favours the formation of calculi by preventing the evacuation of bacteria and also of precipitated cholesterin and bilirubin-calcium. In the absence of such interference infection may continue for years without causing gall-stones.

The disease is much commoner in people of sedentary habits than in manual labourers, and in women is four times as common as in men.

Feeble contraction of the abdominal wall and deficient movement of the diaphragm aid in causing stagnation of bile in the gall-bladder. In this way cholelithiasis is favoured by obesity, pregnancy, abdominal tumours, ascites, chronic cardiac and pulmonary diseases, and tight lacing. Chronic infection of the alimentary tract is of considerable importance, especially the gastro-intestinal catarrh associated with constipation and alcoholism. Appendicitis is closely related to cholecystitis, which later on may lead to the formation of calculi. A very high percentage of cases of visceroptosis are associated with cholelithiasis; the prolapsed abdominal organs drag on the cystic duct and partially occlude it.

The argument that excess of cholesterin in the blood is of importance is not supported by much satisfactory evidence. The increase of cholesterinæmia at each menstrual period may account in part for the much larger number of women than of men affected by the disease, but tight lacing, obesity, pregnancy, and the sedentary life of so many women are

probably sufficient to explain the disparity in the number of those affected in the two sexes.

Gall-stones have been met with in infancy, but are rare before 20, and most cases occur in people over 40 years of age. The incidence is greater in temperate than in tropical regions. The disease is an extremely common one, for gall-stones are found in 7 per cent. of all bodies examined after death.

Pathology.—Calculi may be classified according to their composition:

1. Cholesterin and bilirubin-calcium.
2. Bilirubin-calcium.
3. Cholesterin.
4. Calcium carbonate.

1. *Mixed cholesterin and bilirubin-calcium calculi* are commonest. They may be single or multiple. Single calculi are often large and oval, multiple ones are frequently small and faceted. They are generally dark brown, but are sometimes pale or nearly black, and are soft and greasy in consistence.

2. *Pure bilirubin-calcium calculi* arise in the bile-ducts, and vary in size from that of a grain of sand to that of a pea. Some are brown and rough, others black and smooth.

3. *Pure cholesterin calculi* are single and of slow formation. They arise in the gall-bladder, when the cystic duct is blocked, and are white or yellow and translucent. Some cholesterin calculi contain a little biliverdin or bilirubin and calcium, when they are usually laminated and white and brown or green in colour. They are found under conditions of partial obstruction to the cystic duct, and contain 70–90 per cent. of cholesterin.

4. *Calcium carbonate calculi* are very rare. They are opaque to X-rays.

Calculi may be solitary or may be present in great numbers up to several thousands. The nucleus is usually a collection of bacilli, very rarely a foreign body such as a fragment of roundworm. The majority are formed in the gall-bladder or intrahepatic ducts; those found in the cystic duct or common duct have generally arisen elsewhere. When they are numerous the bile becomes very thick and is sometimes semi-solid.

The walls of the gall-bladder usually become thickened and, if ulceration occurs, local adhesions are formed. If the cystic duct is occluded the whole viscus may be greatly distended and its walls very thin.

Symptomatology.—It is often stated that in many cases gall-stones produce no symptoms, chiefly on the ground that they are very often

GALL-STONES

found at autopsy in cases in which their presence was unsuspected during life. Probably symptoms are produced in the majority of cases, though they are often wrongly interpreted. So long as calculi remain in the gall-bladder and cause no special complication, the symptoms tend to be indefinite or misleading. They cause dyspeptic symptoms, especially flatulence, a feeling of fullness, and dull pain in the epigastrium after food. Sometimes there is dull pain in the lumbar region or under the right scapula, or there may be sharp but slight pain in the right side of the chest. Headache, drowsiness, and slight attacks of shivering may be present, but even these probably indicate a very mild cholecystitis.

Symptoms and signs of a characteristic nature are produced only by inflammation or obstruction and therefore appear in but a small proportion of cases. The symptoms are biliary colic, vomiting, shivering, and collapse, and the signs are tumour, jaundice, and the passage of a calculus in the motions.

Biliary colic.—The most severe attacks of biliary colic are caused by the passage of a gall-stone down the bile-ducts, but definite attacks of colic may be set up by a calculus in the gall-bladder. Colic may also be caused by inspissated mucus or bile pigment in the ducts, and by cholecystitis in the absence of calculi.

The onset of an attack may be quite sudden, or it may be preceded by shivering, nausea, or vomiting, which are probably due to cholecystitis, a condition very favourable to the passage of a stone into the cystic duct.

The pain is excruciating, and the patient rolls about and groans in agony. If untreated, the severe pain may last for some hours, and then suddenly disappear owing to the passage of the stone into the duodenum, or it may last for some days as a dull aching pain with paroxysmal attacks which are almost unbearable. The repeated paroxysms may be associated with the passage of a succession of calculi into the intestine. Attacks may begin at any hour, but perhaps the most usual time is during the night. Pain is generally felt most acutely in the right hypochondrium, and radiates to the epigastric, umbilical, and hypogastric regions. Sometimes it extends to the right shoulder or even down the arms and thighs, and back-breaking pain may be felt at the level of the lower dorsal vertebræ to the right of the spine.

Reflex vomiting accompanies the periods of

acute pain, and the vomit is often bile-stained. During an attack there are constipation and complete anorexia. Rigors or shivering with profuse perspiration are met with during the paroxysm.

There is fever due to cholecystitis in more than half the cases. Leucocytosis and albuminuria are not uncommon. The abdomen is generally rigid and contracted and acutely tender, especially in the right upper quadrant. Dry cough and hiccough sometimes occur.

Enlargement of the gall-bladder, liver, and spleen is said to be a frequent accompaniment of an attack, but is difficult of determination owing to the rigidity and tenderness of the abdominal muscles. Epileptiform seizures have been described in connexion with severe colic, and sudden death has occurred. The pulse is feeble, but often maintains its normal rate or even becomes slower. There may be a transient systolic murmur at the heart's apex. After an attack is over the patient is often greatly prostrated and very drowsy, and may be troubled with dyspepsia and flatulence for some days. The abdominal muscles over the gall-bladder may remain tender for a considerable time.

Jaundice follows in 50–75 per cent. of the cases, but is often very slight. It appears in a few hours or as late as two days after an attack. Many of the slighter cases are due to descending cholangitis and not to obstruction of the ductus choledochus by a calculus. It may develop even though the calculus remains in the gall-bladder or cystic duct.

An unusual complication of biliary colic is intestinal volvulus, which leads to acute intestinal obstruction. Symptoms of obstruction with great distension of the bowel may be produced by ileus due to atony of the intestine. Rupture of the gall-bladder or bile-duct is extremely rare.

The first attack of biliary colic is nearly always the worst. The duct becomes enlarged by the passage of the calculus and by inflammatory changes, so that, although calculi subsequently passed may be much larger than the first, the pain is less severe. The calculus may fail to reach the duodenum and become impacted in the cystic duct or at the ampulla of Vater. In the latter case it is likely to cause intermittent hepatic fever. This complication is discussed later.

Diagnosis.—The indefinite nature of the symptoms produced by gall-stones in the gall-bladder makes diagnosis very difficult. Some-

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times an enlarged gall-bladder can be felt, and, in a few of the cases in which it is full of gall-stones, crepitus can be elicited. Dyspeptic symptoms which do not yield to treatment, in stout middle-aged people, especially women, are often due to gall-stones. If a test-meal proves that the gastric juice is normal and a bismuth meal shows that the muscular action of the stomach is natural, the probability that the symptoms are due to cholelithiasis is increased, but it must be remembered that disease of the stomach and intestine is often associated with cholelithiasis. A past history of enteric or paratyphoid fever is in favour of a diagnosis of gall-stones.

X-rays give little help. Unfortunately, the subjects of this disease are often stout and the gall-bladder lies in contact with a large solid organ, the liver. In addition, gall-stones, with the exception of the rare calcium carbonate calculi, are not very impervious to the radiations. In spite of these difficulties, excellent photographs are sometimes obtained (see PLATE 47, Fig. 5, Vol. III, facing p. 562), but in most cases in which gall-stones are found subsequently by exploratory operation no recognizable shadow is produced, and no weight must be attached to a negative skiagram.

When biliary colic develops, diagnosis is easier. In all attacks of pain suspected to be due to gall-stones the faeces should be passed through a sieve after having been broken up in a solution of carbolic acid; but even in proved cases of cholelithiasis a calculus is found only in about a quarter of the cases.

The differential diagnosis from *cholecystitis* is often impossible, though the pain is usually less severe in non-calculous cholecystitis, and jaundice occurs much less frequently.

In *right renal colic* the paroxysmal pain usually radiates downwards to the right groin, or in a man to the right testicle, and not upwards to the scapula. The urine will be found to contain albumin, blood, and pus. The pain of a *floating kidney* may simulate that of gall-stones, but the presence of a movable tumour can generally be detected. Slight recurrent jaundice sometimes accompanies the attacks of pain in floating kidney and adds to the difficulty of diagnosis. In *duodenal ulcer* the pain may have the same distribution as in gall-stones, but it is much more closely related to food, usually recurring two to four hours after meals and being relieved by taking more food. In this disease hyperchlorhydria is present, whereas it is rare in cholelithiasis. Its

presence may be suspected by the relief of pain after taking bismuth or sodium bicarbonate, or proved by examining a test meal. Occult blood is often found in the faeces by the benzidine test in duodenal ulcer. *Pyloric stenosis* may cause attacks of pain, but the presence of peristalsis and often of a movable tumour at the pylorus, the signs of dilatation of the stomach and the large size of the vomits, usually make diagnosis easy. In *hyperchlorhydria*, attacks of nocturnal pain may give rise to a suspicion of gall-stones, but pyrosis and heartburn are against their presence, and relief of pain by bismuth is in favour of hyperacidity of the gastric juice. Attacks of pain are common in *mucous colitis*, and their severity is often magnified by the neurotic condition of the patient. Tenderness in this disease usually follows the whole course of the colon, and mucus and intestinal casts can be found in the faeces. A *ventral hernia* with omental adhesions may cause attacks of vomiting or pain. Its discovery and the cure of symptoms by operation settles the diagnosis. In *acute pancreatitis* the pain is as severe as in the worst cases of biliary colic. It is more continuous, worst in the epigastrium rather than in the right hypochondrium, and causes more profound collapse. *Lead colic* may give rise to attacks of pain resembling those due to gall-stones, but the occupation of the patient and the presence of a blue line on the gums generally lead to a correct diagnosis. When the severe general pain has gone there is no local tenderness over the gall-bladder in this disease. *Abdominal aneurysm*, especially in the early stages, may be mistaken for cholelithiasis, but is a much rarer condition. Arterial thickening and a positive Wassermann reaction may be aids to correct diagnosis even before a pulsating tumour can be felt. Gall-stones may cause attacks of left-sided pain resembling *angina pectoris*, but they are not relieved by amyl nitrite like true angina, and the size of the heart is normal, whereas in angina it is often large and aortic disease is common. The hepatic and gastric crises of *tuberculosis dorsalis* may resemble biliary colic, but they are relatively uncommon, and the presence of Argyll-Robertson pupils, absence of knee-jerks, and other signs of organic nervous disease should prevent a mistaken diagnosis. In *hysteria* there may be pains like those of cholelithiasis, but there is often a history of other hysterical manifestations, and the age and manner of the patient are seldom those of

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a sufferer from gall-stones. In *spinal neuralgia* there is no vomiting and no collapse; but there is a tenderness over the spine and the skin along the course of the affected nerves.

Complications. Cholecystitis.—As gall-stones owe their origin to mild infection of the gall-bladder, so their presence is liable to keep up a chronic infection which may become acute at any time and cause suppurative, phlegmonous, or gangrenous cholecystitis.

They are also the chief cause of *ulceration of the gall-bladder and bile-ducts*, which leads to adhesions between them and neighbouring organs, and to perforation, or occasionally to hæmorrhage. Healing of an ulcer may produce scarring and sometimes hour-glass constriction of the gall-bladder. *Perforation* may take place into the general peritoneal cavity and cause fatal general peritonitis. This is commonest in the acute phlegmonous or gangrenous cases. When infection is less acute, adhesions are generally present, and perforation occurs into a pocket of peritoneum and causes a local abscess. This may open into an abdominal viscus, usually the duodenum or stomach, or externally through the abdominal wall, or it may cause subphrenic abscess or empyema thoracis. Very rarely the gall-bladder ulcerates into the liver and causes an abscess in its substance. *Fistulæ* originate in this way. They may open externally, usually near the umbilicus; less often there is a communication between the gall-bladder and the gastro-intestinal tract, especially the duodenum. Sometimes there is a fistula between the common bile-duct and the duodenum. *Fistulæ* into the duodenum may lead to duodenal ulceration, hæmorrhage, cicatrization, and even obstruction. Ulceration and obstruction may cause severe vomiting. Broncho-biliary fistulæ are rare. Large quantities of bile, and occasionally calculi, are coughed up. Recovery may be spontaneous, or it may occur after removal of the gall-stones by operation. Other cases are fatal.

Calculus in the cystic duct.—In the cystic duct a calculus may cause inflammation, ulceration, and subsequent cicatrization and adhesions.

Sometimes a diverticulum containing an encysted stone is formed. The contraction of adhesions round the duct or the pressure of an encysted calculus may compress the hepatic or common bile-ducts.

Perforation may cause local abscess or fistula.

Ascending cholangitis is a not uncommon sequel. Impaction, usually near the neck of

the gall-bladder, may occur, and the stone may remain impacted for a long period. If the flow of bile be stopped completely, the gall-bladder becomes greatly distended. The contents at first are mucus and bile, later they become clear and colourless, and the swelling is known as a *mucocoele*. Some mucocoeles become infected by organisms of low virulence, and may form chronic empyemata of the gall-bladder.

Spasmodic contractions of the muscle take place and cause biliary colic, which is seldom so severe as that associated with the presence of a calculus, but which may cause nausea and vomiting. Jaundice is uncommon, but is said to occur in 10 per cent. of the cases.

The distended gall-bladder usually forms a pear-shaped tumour, about the size of a fist. Sometimes it becomes larger and more elongated, and in rare instances may become enormous. As a rule, it lies just below the right costal margin with the larger end downwards, and feels smooth and tense, but when associated with a Riedel's lobe of the liver it may lie in the right iliac fossa. It is not tender unless cholecystitis be present.

A mucocoele is most often mistaken for a floating kidney, but must be diagnosed from hydronephrosis, renal tumour, carcinoma of the pylorus or colon, ovarian cyst and hydatid cyst. In characteristic cases it is freely movable from side to side, but cannot be displaced downwards. Only in rare cases, in which the calculus is not fixed firmly, does it vary in size. If pressed back with the patient recumbent, it rises at once to the front of the abdomen. A *floating kidney* is much more elusive and mobile; generally it can be passed down into the lower part of the abdomen, and when pushed back with the patient lying prone it remains. In the case of *renal tumours* the colon usually lies in front, whereas it lies behind the gall-bladder in tumours of this organ. If distended with air it presses a renal tumour backwards, but pushes an enlarged gall-bladder forwards. Sometimes the colon lies in front of a mucocoele, so that this test must not be relied upon completely.

A *pyloric growth* tends to lie with its long axis across the abdomen, rather than vertical like a gall-bladder: it is harder, and tenderness is more often elicited than in the case of a distended gall-bladder. Gastric symptoms are more prominent in pyloric tumour and dilatation of the stomach is often recognizable. *Carcinoma of the colon* also generally causes a

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swelling which lies transversely; it is not smooth and tense like a mucocoele, but is more indefinite, and, if the lumen of the gut is narrowed, it varies in size according to the amount of faecal matter lying behind it. Enemata often reduce its size.

An *ovarian cyst* seldom causes difficulty in diagnosis unless the pedicle be long and the cyst adherent. An *hydatid* near the anterior border of the liver may be impossible to differentiate without a laparotomy. Exploration by aspiration is not permissible. Eosinophilia, if present, is much in favour of hydatid disease.

Calculus in the common bile-duct.—A stone in the ductus choledochus may cause infective cholangitis (intermittent hepatic fever), ulceration, perforation with local abscess-formation, suppurative cholangitis, and hepatic abscesses.

Intermittent hepatic fever.—In this condition the stone is usually impacted near the ampulla of Vater and gives rise to a train of very characteristic symptoms. In part these are caused by ascending infection from the duodenum or from the gall-bladder, in part from the mobility of the stone in the duct, in a dilatation of which it lies loosely and acts as a ball-valve. During exacerbations of the infection the mucosa swells and the duct is almost completely obstructed, but in the intervals bile trickles past. The condition may begin many years after the first formation of gall-stones, and the original symptoms may have been so slight as to have given rise to no suspicion of their presence.

During the periods of acute cholangitis there are fever, rigors, sweating, and pain, often of great severity, in the epigastric and hepatic regions. There are also vomiting and gastric pain. Leucocytosis is present, and the spleen is often enlarged. Jaundice may be absent, more usually it is present and may become intense. The liver is enlarged and tender, but the gall-bladder is seldom palpable. There is nearly always some interstitial pancreatitis, which may account for the glycosuria in some of the cases. Urobilin is present in the urine. In the intervals the signs and symptoms may disappear entirely, or some enlargement of the liver with slight jaundice may persist. Recurrent attacks may go on for many years.

Suppurative cholangitis is the commonest complication, and may lead to suppurative pancreatitis, hepatic abscess (multiple or solitary), or pyelephlebitis. Ulceration of the bile-duct with obliteration of its lumen by cicatrization is rare. In longstanding cases ob-

structive biliary cirrhosis may result.

Diagnosis of intermittent hepatic fever.—The attacks may be mistaken for malaria, but in malaria jaundice is uncommon and never deep, nor does it persist in the quiescent intervals. Splenic tenderness and anæmia are more typical of malaria, and parasites will be found in the blood during an attack. In *suppurative cholangitis* the condition of the patient is much worse, the fever continuous, and the liver larger and more tender; the gall-bladder may be palpable, and the jaundice is lighter but constant if it be present at all. *Malignant disease of the liver* causes more rapid wasting. Pyrexia, if it be present, is lower and not periodic. The liver is generally enlarged and nodular. Malignant growth pressing on the bile-duct or in the head of the pancreas causes deepening jaundice and complete disappearance of stercobilin from the fæces. There is rarely any fever, and the gall-bladder is generally distended.

The following diagnostic table is useful in differentiating calculus from carcinoma of the pancreas:—

	Calculus	Carcinoma of pancreas
Cambridge's pancreatic reaction	Present in 66 per cent. of cases	Absent in 66 per cent. of cases.
Urobilinuria	Present	Rare.
Crystals of calcium oxalate	Present	Rare.
Fat in fæces	Proportion of saponified exceeds unsaponified	Proportion of saponified less than or equal to unsaponified.

In chronic *simple cholangitis* the pain and fever are less marked, and rigors and sweats are generally absent. *Hypertrophic biliary cirrhosis* causes much greater splenic enlargement and the exacerbations are less severe than in intermittent hepatic fever; in the intervals jaundice is deeper in cirrhosis, whilst splenic enlargement disappears in intermittent hepatic fever in the latent periods.

Prognosis in intermittent hepatic fever.—Owing to the risk of suppuration and of degeneration of the liver-cells the outlook is bad in well-marked cases if no operation is undertaken. If operation is performed before too much damage is done to the liver and pancreas the results are generally good. In neglected cases chronic pancreatitis may cause complete obstruction to the bile-duct and eventually prove fatal.

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Treatment of intermittent hepatic fever.—If attacks are infrequent it is well to try medical treatment first in the hope of the calculus reaching the duodenum and the cholangitis subsiding permanently. During quiescence a light diet and purgatives are necessary. Carlsbad water at home or a course of spa treatment can be tried. The best medicines are urotropine and sodium salicylate. *B. coli* vaccine is sometimes useful. In cases with frequent attacks operation for removal of the calculus must not be delayed too long; two months' delay is permissible.

During acute attacks the pain may be relieved by exalgin or antipyrin. Morphine may be needed, but should not be given unnecessarily. Local treatment for vomiting and dyspeptic symptoms is of little use, as they are for the most part reflex in origin.

Malignant disease.—Calculi in the gall-bladder and common bile-duct are wont to cause carcinoma. They are met with in 70 per cent. of all cases of carcinoma of the gall-bladder and in about 30 per cent. of cases of carcinoma of the bile-ducts. These percentages show the intimacy of the association.

Prognosis in cholelithiasis.—Gall-stones in the gall-bladder, in the absence of complications, make little or no difference to the duration of life, and the symptoms are often slight. In biliary colic immediate death from vagal inhibition is extremely rare, and the dangerous complications of perforation, volvulus, or ileus are very uncommon. Medicinal treatment often gives excellent results, while after surgical interference gall-stones seldom form again. If a smooth oval stone is found in the faeces after an attack of colic, it is probable that there will be permanent freedom from further symptoms; but if a faceted calculus is discovered, other attacks can be foretold with some certainty.

Cholecystitis often yields to treatment, but there is always a risk of the development of the acute suppurative or phlegmonous forms, which are dangerous to life.

The dangers of impaction in the cystic or common bile-duct have been dealt with already. In both conditions, if medical treatment fails, timely surgical interference is generally successful. Most of the other complications are curable, but suppurative cholangitis, pancreatitis, and pyelephlebitis are very fatal.

In spite of the severity of the surgical operations necessary in some cases, and of the

fact that many of the patients are not good subjects for operation, the mortality in skilful hands is comparatively low. Moreover, only a small proportion of the sufferers from cholelithiasis develop complications requiring an operation.

Treatment.—The treatment must be both local and general. Certain sodium salts—the chloride, bicarbonate, benzoate, sulphate, salicylate, and phosphate—have been accredited with the power of dissolving gall-stones *in situ*, but it has been proved that no solvent action is produced *in vitro* by a 1-per-cent. solution, and they cannot be supposed to act more efficaciously in the body.

Olive oil undoubtedly dissolves stones in a test-tube, but it is impossible for the oil to reach the gall-bladder. The favourable results reported are based on the discovery of supposed calculi in the faeces after the administration of large doses. They are really compounds of calcium with oleic, palmitic, and margaric acids, and there is no record of a true gall-stone being found after olive-oil treatment. It is apt to cause dyspepsia, and probably does more harm than good.

The only rational treatment of this kind is to increase the amount of bile-salts in the bile. Sodium glycocholate is a solvent of cholesterin, and bile is the most efficient cholagogue we possess. Bile-salts may be given alone ($\frac{1}{2}$ –2 gr.), or as *fel bovinum* in salol-coated pills or capsules of 5 gr. each, in doses up to 15 gr. three times a day.

Biliary antiseptics such as sodium salicylate and urotropine are useful for cholecystitis or cholangitis. If these drugs fail, a vaccine of *B. coli* is sometimes valuable.

Massage has been recommended to aid in expelling calculi, but if ulceration is present there is danger of causing perforation, and this form of treatment should never be employed.

The most important points in **general treatment** are regulation of the diet and exercise. The diet must be moderate in amount and simple in character. All rich and irritating foods should be avoided. Fats, especially animal fat, should be restricted, and carbohydrates, such as sugar, bread, and potatoes, should be taken sparingly. Fresh green vegetables, salads, and fruit are useful. Light wine is quite allowable.

For sufferers from gall-stones who are obese or lead sedentary lives, regular exercise is essential. Walking part of the way to and

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from business, and golf at the week-end, promote the flow of bile. For women with sedentary habits, daily walks must be insisted on; tight lacing should be avoided.

Spa treatment is very valuable. In the United Kingdom, Harrogate and Llandrindod Wells have the highest reputation; and abroad, Carlsbad, Vichy, Marienbad, Homburg, Neuenahr, and Bertrich can be recommended. For those who cannot afford the necessary time or money, home treatment can be carried out with excellent results. A glass of hot alkaline water should be taken before breakfast, followed by half an hour's walk; this is to be repeated in the afternoon, and a third glass of hot Carlsbad or Vichy water should be taken at bedtime. If the patient remains constipated, Apenta water or Hunyadi János should be taken at meals and a pill (calomel $\frac{1}{4}$ gr., pulv. rhei. co. 4 gr.) every night or as often as is necessary.

During acute attacks of biliary colic nothing is of use except a hypodermic injection of morphine, and inhalation of chloroform until the morphine has had time to act. Draughts of sodium bicarbonate in hot water sometimes help to relieve pain and vomiting. In slighter cases $\frac{1}{2}$ gr. of exalgin every half-hour for four doses, or a draught of spiritus etheris in two teaspoonfuls of chloroform water given every quarter of an hour, may be sufficient. Hot fomentations should be applied to the liver region.

Operative treatment should not be undertaken during an attack, unless some urgent complication is suspected. The medical treatment of cholelithiasis should give place to surgical if the condition of the patient does not improve after fair trial. Medical treatment may fail owing to adhesions, obliterative cholecystitis, the presence of a simple empyema or some other complication.

Special indications for surgical interference are: (1) Recurrent attacks of biliary colic; (2) intermittent hepatic fever; (3) tumour of the gall-bladder due to a large collection of gall-stones, mucocele, or empyema; (4) severe gastric symptoms which do not yield to treatment: these are probably due to adhesions; (5) persistent jaundice; (6) interstitial pancreatitis; (7) suppurative, phlegmonous, or gangrenous cholecystitis; (8) intestinal obstruction; (9) fistula; (10) peritonitis; (11) local abscess.

The choice of operation cannot be made until the exact condition has been revealed by open-

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ing the abdomen. Cholecystotomy, cholecystectomy, choledochotomy, and cholecystenterostomy are all useful at different times, but the procedure to be adopted must be left to the discretion of the operating surgeon.

E. A. COCKAYNE.

GALVANISM (see ELECTRICAL TREATMENT).

GANGLION. — A cyst, containing mucoid material, which develops in the substance of the capsule of a joint, in a tendon-sheath, or more rarely in the tendon itself. It is not a protrusion of synovial membrane, as was formerly supposed. Ganglia are met with most frequently on the dorsum of the wrist, but they are also seen in the region of the ankle and the knee. They occur more often in women than in men, and seem to result from prolonged slight overstrain of a tendon or joint. Especially are they seen in pianists, typists, and women who wring clothes. They are accompanied by a sensation of weakness; sometimes by neuralgic pain. The smooth, rounded swellings may fluctuate, but often are too tense to give this last sign, and are then very hard. Their range of mobility depends upon whether they are attached by a broad base or a narrow pedicle. Those at the back of the wrist frequently become fixed when the fingers are clenched.

Treatment.—If there are no subjective symptoms the ganglion may be left alone. When weakness, or pain, or the unsightliness is complained of, the swelling must be got rid of. Many can be made to disappear by a comparatively simple procedure, though success cannot be guaranteed. A tiny incision is made in the overlying skin with a sharp knife under local anaesthesia; through a small puncture in the cyst-wall the mucoid contents are then expressed. With a hypodermic syringe 3-5 min. of pure carbolic acid are introduced into the evacuated cavity and massaged into contact with the whole wall. A dressing is then applied. After some reaction the swelling disappears. If this fails, or if the patient does not want to run the risk of its possible failure, the ganglion should be dissected out. The whole wall must be removed, or recurrence will take place. Every antiseptic precaution must be available, as a joint may be opened. Local anaesthesia should never be used. The ganglion may be adherent to a joint capsule which has not been rendered insensitive, and the surgeon be

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tempted to leave this part of the wall because of the pain dissection causes. Recurrence will then be certain.

Removal of a ganglion should not be undertaken unless it can be done under the most favourable aseptic and antiseptic conditions. A longitudinal incision is made over the swelling, which is freed all round and as near the attached area as possible. The swelling is then incised and the mucoid material expressed. The sac wall being pulled upon, free access is gained to the pedicle and the whole sac removed. In doing this a tendon-sheath or a joint-capsule may be opened. A single catgut suture will suffice to obliterate this aperture. The skin is then sewn up. C. A. PANNETT.

GANGLIO-NEUROMA (*see* NEUROMA).

GANGRENE OF CORD (*see* UMBILICAL INFECTION IN THE NEW-BORN).

GANGRENE OF THE EXTREMITIES (including Gas Gangrene). **Etiology.**—Massive necrosis of the extremities is seen much more commonly in the lower than in the upper limb. The actual death of the tissues is due to an interference with their local blood supply, to a lessened power of the tissue-cells to utilize the nutritive material brought to them, or to both these factors in combination. General weakness and poor cardiac action are predisposing causes.

Certain clinical groups based upon the causal agents are recognized :

1. Gangrene due to trauma.
2. Gangrene due to disease of the arterial wall.
3. Gangrene due to arterial embolism or thrombosis.
4. Gangrene due to arterial spasm.
5. Gangrene associated with disease of the central or peripheral nervous system.
6. Gangrene due to physical and chemical agents.
7. Gangrene due to bacterial infection.

Pathology.—The dead tissue is dry, withered and shrivelled up (*dry gangrene*), or swollen and oedematous (*moist gangrene*).

Dry gangrene occurs in a limb whose circulation has been very much curtailed before the onset of the catastrophe. The blood supply, usually through arterial disease, has been gradually diminishing and perilously approaching the border line between the barely adequate

and the insufficient. Local death in the limb is precipitated by the complete breakdown of the vascular supply, consequent on the inability of the circulation to adapt itself to the slightly abnormal conditions brought about by some quite trivial injury. After the death of the tissues, still further loss of water from them takes place by evaporation. The part is thus shrunken, wrinkled, and hard. In colour it is brown, black, or dark yellow. There are no blebs or blisters. It has a musty smell but does not stink.

Moist gangrene is seen where the tissues are engorged at the time of local death and subsequently evaporation is prevented. The dead portion of the limb is swollen (not shrivelled), soft and boggy. Large blebs form on its surface. It is purple at first, then brown, black, and mottled; sometimes it is green. Usually the dead tissue does not remain aseptic, but infection of various degrees of severity comes on. Liquefaction of the tissues then occurs and the products of decomposition (sulphuretted hydrogen, valerianic and butyric acids, indol, skatol, ammonia) make their presence evident by an evil smell. Collections of gas in such circumstances may also be seen.

In the zone of living tissue immediately abutting on the dead mass, should the patient survive sufficiently long, a reaction is set up, which results in the formation of a barrier of granulation tissue. This is evident on the surface as a red line of demarcation. In favourable circumstances this barrier limits the gangrenous process, and the line of demarcation becomes a groove of ulceration which gradually deepens until the dead segment is separated off. After this spontaneous detachment of a necrosed extremity of a limb the stump is found to have a conical shape. The bone projects beyond the soft parts. In the gangrenous limb there is at first a numb feeling, but this is succeeded by severe pain which is very exhausting and prolonged. The limb is cold, and there is a loss of motor power. No pulsation can be felt in the vessels.

The general symptoms attributable to the local conditions in dry gangrene are slight. In moist gangrene symptoms of septicæmia of varying degree are present, or septicæmia may supervene.

1. TRAUMATIC GANGRENE OF THE EXTREMITIES

This variety of gangrene follows either injury to or ligation of arteries (indirect traumatic gangrene), or a large segment of the

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limb may die from a direct crush (direct traumatic gangrene). After ligation of an artery an adequate collateral circulation may fail to become established should the vessel-walls be narrow and inextensible from arterio-sclerosis, should the heart's action be weak, or should the available blood have been diminished by a previous severe hæmorrhage. There are certain "dangerous" arteries, the ligation of which is prone to be followed by gangrene. They are the common femoral and popliteal in the lower limb and the brachial in the upper limb. Ligation of the subclavian only rarely leads to gangrene. Subcutaneous rupture of the main artery of a limb or of an aneurysm of a main artery is a very serious accident. The accompanying vein is usually compressed by the clot, thereby throwing an additional strain on the already crippled circulation. The gangrene following ligation of an artery is frequently dry, because conditions necessitating this operation usually occur in the subjects of arterio-sclerosis. Direct traumatic gangrene and gangrene due to the rupture of a normal artery or subcutaneous rupture of an aneurysm are moist.

Treatment.—When, after an injury, a portion of a limb is so crushed that recovery of the injured part is impossible, or when such damage is done to its vascular supply that gangrene is certain to result, amputation should be performed above the crushed area or at the level of the damage to the main blood supply, immediately the general condition of the patient will admit of it.

When after ligation of an artery there are the most remote grounds for supposing that gangrene may supervene, immediate steps should be taken to anticipate and, if possible, prevent this event. The circulation should be assisted by raising the limb on pillows and wrapping it up in cotton-wool to maintain its temperature, whilst it should be shaved and scrupulously cleaned before swathing it in its wrapping of sterilized gauze and wool. If gangrene should come on and the process follow an aseptic course, amputation should not be done until the line of demarcation manifests itself. The appearance of sepsis renders amputation without delay imperative.

The choice of the site for amputation is determined by the condition of the arterial walls. If the arteries are tolerably healthy the limb may be severed as near to the line of demarcation as is convenient. But for guidance in other cases reference should be made

to the rules laid down for the treatment of senile gangrene.

2. GANGRENE DUE TO DISEASE OF THE ARTERIAL WALL

The arterio-sclerosis of old people may lead to senile gangrene, but a similar form also occurs in much younger persons. Senile gangrene affects the toes more than the fingers, and its onset is determined by some slight and often unnoticed trauma, such as the pressure of an ill-fitting boot, or an injury inflicted in cutting a corn. An inflammatory exudation into the tissue interstices occurs at the site of this small injury, which brings about local capillary stasis by pressure. The vessels are so diseased that they are unable to dilate sufficiently to re-establish the circulation through the damaged part. By a similar process the first small necrotic area may increase, for it causes an inflammatory reaction in its vicinity. Senile gangrene is of the slowly progressive kind, and it is difficult to predict when the mortification of tissue will cease to spread, one line of demarcation being followed by another farther up the limb.

Certain **prodromal symptoms** are common. The limb becomes cold and cyanotic. A sensation of walking on wool may be caused by numbness of the feet. Attacks of pain in the limb are also felt, and a limp appears. The prodromal symptoms are relieved by rest. The actual gangrene shows itself as a black patch on one of the toes. Sometimes a line of demarcation forms, the slough is cast off, and the resulting ulcer heals. Only too commonly the process gradually spreads to the foot and leg as indicated above. The gangrene is usually of the dry type. The pain varies much in intensity, but is apt to be very severe, so that the enfeebled patient becomes worn out by want of sleep. Sepsis supervening makes his condition a precarious one.

In *diabetic gangrene*, not only are the tissues starved of blood owing to the accompanying arterio-sclerosis, but they are unable to utilize such nutritive material as is brought to them. The gangrene, beginning as a dry type, soon becomes moist owing to infection. Organisms appear to grow more freely in the diseased tissue of the diabetic. Mortification in such cases spreads more rapidly, and is a more serious affection than senile gangrene. (*See DIABETES MELLITUS.*)

Syphilitic arteritis occasionally causes gangrene of an extremity.

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Treatment.— When the prodromata of senile gangrene make their appearance the patient should be warned of his hazardous condition. Sufficiently warm socks should always be worn, and the boots must be roomy and not chafe. Corn-cutting must be interdicted. The patient should avoid getting his feet wet and chilled; neither should he expose them, when cold, to the heat of a fire.

When gangrene has declared itself, the part, to a considerable distance above the dead area, must be shaved and wrapped in a dry sterile dressing. The patient is confined to bed, his leg raised on a pillow, and warmth applied by hot-water bottles outside the thick dressing.

General stimulant and supporting treatment should be adopted, and, should hypostatic pneumonia threaten, the patient must be got out of bed and allowed to sit on a chair with the limb raised on a support. Analgesics may be required for the pain. If pain is not intolerable, and if the dead area is small and superficial, no active local measures need be taken. The small slough will perhaps separate and the resulting ulcer heal over. This is a long process.

Amputation is indicated when (1) the pain is severe and unbearable, (2) the condition is advancing, or (3) a whole toe is dead.

No patient should be allowed to become exhausted and enfeebled from continuing pain; nothing is gained by waiting when the process is spreading.

The site for amputation depends very little upon the situation of the line of demarcation. There is only one essential: it is that the amputation be performed through tissue with a sufficiently adequate circulation. Amputation of a toe is nearly always followed by an extension of the gangrenous process upwards from the site of operation, and this is true generally of operations performed in the foot and the region of the lower leg. The lowest situation where operation is free from this risk of gangrene in the stump is the lower third of the thigh. Drastic though such advice appears when only a single toe is necrosed, it is a sound recommendation to follow. An old person, enfeebled perhaps by chronic Bright's disease, exhausted it may be by weeks of pain, is in no fit condition to withstand several operations, and his best interests are followed if an immediate high amputation through the lower third of the thigh is done.

A method of determining the lowest site

where amputation may be practised without fear of necrosis of the flaps occurring, has been employed. The patient being recumbent, the affected limb is raised, emptied of blood, and a tourniquet applied high up the thigh for a few minutes. In a normal limb, when the constrictor is removed, the blush of the filling arterioles of the skin travels downwards at a uniform rate from the site of constriction to the toes. But when the vessels of a limb are affected with arterio-sclerosis the advance of the blush is seen to be checked at a certain level below the obstruction to the circulation. Beyond this the hyperæmia does not advance regularly; the limb here shows for some minutes areas of redness interspersed with white anæmic spaces. Amputation through this mottled area is unsafe, but it can be done through the uniformly hyperæmic area with impunity. By using this method it is possible sometimes to amputate safely below the knee. It is said that the method is not without danger when the arteries are very sclerosed and brittle. Direct damage to the main vessel may be done by the constrictor.

Another method of determining where amputation may be performed safely has been recommended. The main vessel of the limb is exposed and examined by direct inspection. Operation may be done at the lowest level where blood is being freely conveyed. No success has followed the formation of an arterio-venous anastomosis in the thigh for senile gangrene.

In the treatment of diabetic gangrene it is safer to follow the same rule as in senile gangrene and amputate in the lower third of the thigh. It is easy for an infection to become established in these cases, and poorly nourished flaps will certainly conduce to it. Amputation must not be delayed when there is infection in the gangrenous part.

Spinal anæsthesia must be employed. The general treatment to reduce the glycosuria should of course be carried out energetically.

3. GANGRENE DUE TO ARTERIAL EMBOLISM OR THROMBOSIS

Most frequently the embolus is a detached vegetation from a heart affected with endocarditis, or a portion of bloodclot from an aneurysm. It lodges particularly where a large artery bifurcates, as for example in the lower end of the popliteal. A characteristic feature is the sudden acute pain felt at the site of the block. The distal part of the limb

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immediately becomes pale and the signs of local death rapidly appear.

If the arteries are healthy and the obstruction is not at once complete, the gangrenous process is moist in type. If there is arterio-sclerosis it is dry.

In thrombosis, whether due to clotting in an aneurysm or to an inflammatory process spreading to the wall of a neighbouring artery, the pathological change is more gradual. Preliminary muscular cramps, numbness, and tingling may give warning of the approaching condition. The gangrene is dry in type.

Treatment.—The principles of treatment of gangrene due to these causes are the same as after ligation of an artery (*see p. 505*).

4. GANGRENE DUE TO ARTERIAL SPASM

The gangrene from chronic ergotism following ingestion of infected rye bread belongs to this group, but is almost unknown in these islands. The lower limb is most often affected, but the fingers may be attacked, and even the tips of the ears. The general symptoms of chronic ergot poisoning are present: weakness, headache, tinnitus, disturbance of vision, vomiting, and diarrhoea; the gangrene is dry.

In Raynaud's disease some derangement of the vaso-motor mechanism is responsible for a sudden, usually symmetrical, spasm of the arteries of the upper or occasionally of the lower limbs. Cold is particularly liable to call forth this spasm, but the exciting cause may be some mental shock, and the patients are often of an hysterical temperament. Very rarely Raynaud's disease comes on in exophthalmic goitre. It is not often seen in men. In the case of the upper limb there are numbness and pain in one or more fingers, which become pallid; this gives way after a time to a dusky cyanosis. The circulation may become re-established and the fingers return to normal. Should the spasm last beyond the viable period of anæmia, recovery is incomplete and necrosis occurs, but the slough is nearly always superficial. On the black area a vesicle appears, which dries up, forming a crust on the tip of the finger. Sometimes the necrosis is more extensive. The separation of the slough is very slow, and takes weeks or months. (*See also RAYNAUD'S DISEASE.*)

A form of gangrene of the fingers, which although uncommon is avoidable, must be referred to here. Operations upon the fingers are so frequently performed by conduction anæsthesia, a circle of anæsthetic being injected

round the base of the finger, that it is necessary to bear in mind that adrenalin added to the local anæsthetic agent may lead to disastrous results. The spasm of the four digital arteries may last long enough to determine gangrene of the extremity of the finger. There is no need for the use of adrenalin, inasmuch as the anæsthetic may be kept in the vicinity of the nerve-trunks by the simple device of surrounding the base of the finger with an elastic band.

Treatment.—In Raynaud's disease amputation is seldom called for. The superficial sloughs separate and the ulcers gradually heal. Should amputation be necessary, however, it may be done just above the necrotic area.

5. GANGRENE ASSOCIATED WITH DISEASE OF THE CENTRAL OR PERIPHERAL NERVOUS SYSTEM

Gangrene of this type in the limbs usually takes the form of limited death of tissues, and does not always begin in the extremities of the fingers or toes. Several factors play a part in bringing about the condition. Anæsthesia prevents the patient being apprised of noxious stimuli and so deprives him of the opportunity of counteraction. Vaso-motor changes and the absence of trophic impulses help to render the tissues more vulnerable. The underlying nerve condition may be a hemiplegia, paraplegia, syringomyelia, locomotor ataxia, peripheral neuritis as in leprosy, or the complete traumatic severance of a nerve-trunk. Spina bifida, or the growth of a tumour in the spinal cord or peripheral nerves, also leads sometimes to localized gangrene of the extremities.

Examples of this type of gangrene are the bedsores on the heels of patients suffering from paraplegia and the trophic ulcers of the sole seen in tabes dorsalis.

Treatment.—For the treatment of trophic ulcers, *see* ULCERATION. Massive gangrene will require amputation.

6. GANGRENE DUE TO PHYSICAL AND CHEMICAL AGENTS

Gangrene due to the extremes of heat and cold, to strong electric currents, and to exposure to the X-rays and radium is considered elsewhere (*see* BURNS AND SCALDS; FROSTBITE; DERMATITIS, X-RAY).

Carbolic-acid gangrene must be referred to here, and a warning issued against the use of a carbolic-acid dressing for the fingers and

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toes, for a number of cases have been reported where it has been followed by gangrene. Of all applications, a carbolic fomentation is fraught with most danger, for here volatilization of the acid is prevented by a covering of impervious material. Even a 1-per-cent. solution has caused gangrene. Moist lysol or alcohol dressings applied to the fingers have also led to the same disastrous results, but the risk with them is not so great as with carbolic acid. They should all be eschewed as antiseptics for fingers or toes.

When gangrene has occurred, amputation should be done as soon as the line of demarcation appears, and immediately above it.

7. INFECTIVE GANGRENE (GAS GANGRENE)

This terrible condition had passed beyond the purview of the older surgeons, and was not within the ken of the majority of the newer generation, when in 1914 it appeared again on the battlefields of France; a useful reminder that preventive measures against banished infective diseases can never be finally discarded.

Etiology.—The infective organisms of gas gangrene are the *Bacillus perfringens* (*B. aerogenes capsulatus*, *B. welchii*) and the *Bacillus sporogenes* (*B. maligni oedematis*). Experiment has shown that the injection of these micro-organisms alone is not sufficient to initiate the condition, certain contributory factors being necessary, viz., simultaneous infection with other organisms, the absence of a free supply of oxygen at the site of infection, gross local tissue injury, or greatly lowered general resistance due to hæmorrhage, shock, or extreme fatigue. Serous cavities have a higher resistance to these anaerobes than have the muscles.

The **clinical features** vary in different cases. The gangrene may be localized, it may be confined at first to a single muscle or muscle group, it may be a slowly spreading or a fulminating process. In the last type of the disease it is very formidable, the gangrenous process spreading from leg to umbilicus or from forearm to axilla in a few hours. Locally, the characteristic feature is the production of gas in the tissues; the whole region is swollen, the skin is red, and pain is severe. Resonance to percussion may be obtained. Very soon the part dies; the colour changes from brick-red to green and black, and blebs filled with fluid and gas appear. The tissues liquefy and give forth an unbearable stench. The limb becomes a soft, liquid- and gas-exuding, foul-smelling,

shapeless, crepitating mass. The general symptoms due to the absorption of poisonous products are extremely grave. They are pallor of the face with cyanosis of the lips, a running feeble pulse, a primary rise of temperature to 102°–103° F. succeeded by a drop to the subnormal, general apathy, and vomiting of altered blood. If the disease be not checked, death follows, due to the toxæmia. Sometimes a gas-gangrene septicæmia appears and lasts for a few weeks; rarely a pyæmia. Both these conditions are fatal.

Treatment must be undertaken without delay. Shock must be combated by every possible means. Hæmorrhage, if it be still proceeding, must immediately be stopped. If the local treatment cannot be undertaken at once, or the general condition is too bad for the necessary surgical measures to be proceeded with, an effort should be made to procure sleep and the patient should be induced to take nourishment. Sodium bicarbonate and glucose (5 per cent.) should be administered by rectum or given by mouth. Immobilization of the limb is very important. A wet Dakin dressing should be applied. The local measures to be adopted depend upon the clinical type assumed by the disease.

When it is a slowly spreading local lesion the wound must be excised and treated by the Carrel-Dakin method. Incisions should be made down to and through the deep fascia in the neighbouring parts well beyond the limits of spread. It is essential also to remove every particle of foreign matter in the wound.

When the disease is confined to a muscle or group of muscles, these must be excised in their entirety. The resulting wound is treated by the Carrel-Dakin method.

When the disease is advancing, not a moment is to be lost. Amputation is urgently called for. If the blood-pressure has not already fallen too far, the lower extremity may be removed under spinal anæsthesia. In the case of the upper limb, or when the blood-pressure is low, gas and oxygen should be the anæsthetic used. The operation should be performed rapidly, through normal tissue if possible, but it must be done even though the process has extended to the trunk, in which case the diseased area above the amputation has incisions made into it. A guillotine operation is recommended by some surgeons, but flaps may be fashioned. The essentials are that (1) the amputation be done rapidly, (2) that as little hæmorrhage as possible

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take place, (3) that the flaps be left widely open, and not a single stitch inserted, and (4) that the widely open wound be treated by the Carrel-Dakin method (*see under WOUNDS, TREATMENT OF*). Usually a second operation is necessary to fashion a useful stump. C. A. PANNETT.

GANGRENE OF LUNG (*see LUNG, GANGRENE OF*).

GANGRENOUS APPENDICITIS (*see APPENDICITIS*).

GANGRENOUS PANCREATITIS (*see PANCREATITIS, ACUTE*).

GANGRENOUS STOMATITIS (*see STOMATITIS AND GLOSSITIS*).

GAS GANGRENE (*see GANGRENE OF THE EXTREMITIES*).

GAS POISONING OF WAR, AFTER-EFFECTS OF.—This article deals only with the later symptoms and signs which may follow, or be alleged to follow, as a result of "gassing."

In estimating the truth or otherwise of the assertion that such disabilities are due to the effects of gas, attention must first be paid to the nature of the gas to which the sufferer claims to have been exposed. This point is often difficult to ascertain; the patient may be completely ignorant of its nature, and exposure to several kinds of gas may have occurred at the same time. In such cases the severe symptoms of asphyxiating or irritant gas may have overshadowed the apparently slighter but more lasting effects of a simultaneous exposure to a gas such as phosgene.

The history of the early effects is, however, of value. The irritant gases such as "mustard" and "tear gas" produce severe irritation of the skin or mucous surfaces, and in the former case blistering of the skin and intense conjunctivitis will probably have occurred, the blistering being often associated with, or followed by, deep pigmentation of the skin surface. Phosgene gas produces no such effects upon external surfaces; its action is that of a powerful cardiac and nervous depressant.

After exposure to gas in explosive mines a definite and reliable history is usually obtainable. The harmful gases in such cases are carbon monoxide and nitrous fumes. Their immediate symptoms are circulatory and physical depression, with, in the case of nitrous

fumes, some respiratory irritation. These effects are not lasting, and exposure to these gases is unlikely to be claimed as the cause of late or persistent disability.

Symptomatology.—The symptoms which are likely to be ascribed by the patient to exposure to gas are *cardiac, respiratory, and nervous*. The previous history of the patient with special reference to the system involved must be very carefully investigated; if in a previously healthy man there is a definite history of symptoms which can be attributed to the effects of gas arising shortly after he has been so exposed, it is justifiable to lay the blame on the gas.

Circulatory system.—Disordered action of the heart is very commonly complained of. It is too early to form a reliable opinion as to its possible duration. Præcordial pain is very frequent; it is usually localized to an area below the left nipple, but may be much more extensive; it may be constant or, as is much more usual, be induced by exertion or excitement. Its occurrence, especially when associated with skin or muscle tenderness, is to be regarded as evidence that the heart-muscle is distressed, but is not necessarily a sign that the heart is diseased. Palpitation coming on apparently spontaneously, or on slight exertion, may be cardiac in origin; more often it is due to a nervous defect. Breathlessness, especially at night, waking the patient from sleep or preventing sleep while lying flat, is, in these cases, usually a sign of cardiac distress. Dilatation of the heart may occur: it gives rise to the usual signs and symptoms, but as the cause which has produced it has been only temporary in its action, its prognosis in an otherwise healthy heart is good. Among several thousand gassed patients seen by the writer there has been no case of fresh endocarditis. Localized peripheral gangrene occurs in a proportion of severe cases. It would appear reasonable to expect that vessels which have been damaged by the effects of gas may later be more prone to degenerative change than healthy vessels, but no such case has yet been described.

The *treatment of the cardiac disabilities* differs in no particular from that of corresponding disabilities due to other causes. Their prognosis in view of the non-continuance of the exciting cause is good.

Respiratory system.—The intense tracheitis and bronchitis which result from asphyxial gas-poisoning are often associated with definite

destruction of mucous surfaces. The resultant changes are slow in recovery, and a tendency is left to severe reaction to slight irritation. Serious capillary bronchitis and broncho-pneumonia were especially frequent complications of influenza in such cases during the epidemic of the autumn of 1918.

Chronic bronchitis, paroxysmal cough, and the development of emphysema may result from severe gas-poisoning. Persistent aphonia is not uncommon. It owes its origin to definite local irritative lesions, but its permanence is apt to be encouraged by over-treatment. If careful local examination fails to reveal a sufficient cause for its presence, treatment by persuasion should be carried out.

Attacks of nocturnal dyspnoea are occasionally complained of. Some of these appear to be of the nature of true asthma and are possibly due to reflex stimulation from a focus of irritation in the upper respiratory passages resulting from their exposure to gas. Others, occurring more especially after exposure to phosgene, are more in the nature of "cardiac asthma." Treatment of the condition should be carried out on the usual lines, any possible source of irritation being searched for and dealt with locally.

It has been shown that prolonged exposure to an atmosphere containing a high percentage of oxygen has produced beneficial results, but this method of treatment is impracticable, and its ultimate results are still uncertain.

As a result of gas-poisoning and its consequent bronchitis a quiet focus of tubercle in the lung may be stirred into activity. In my experience such cases do badly.

Nervous system.—The nervous symptoms ascribed to gassing are mostly subjective. Numbness of one or more limbs may be complained of; headache, usually frontal or vertical, is common; sleeplessness and emotional instability are often troublesome. The most constant symptom is a lassitude of mind and body, which may last for months.

Some disorder of the eyes is often mentioned; the eyes are easily fatigued, and night-blindness may be complained of. Blepharospasm is common; if no local lesion to account for this is present, stimulating treatment, such as douching the eyes forcibly with cold water, is indicated. The use of atropine and eye-shades is to be discouraged.

The man who has been severely gassed must be regarded as one who has survived

the equivalent of a severe illness. His convalescence may be slow, and complete recovery may be delayed more in one system than in another. The results of the changes which have occurred may be none the less lasting and none the less real because they are sometimes indefinite. The psychical element in the patient's condition is difficult to gauge. His treatment is not altered because his disability dates from a gas attack, and his prognosis is uniformly good. C. E. SUNDELL.

GASTRALGIA (see STOMACH, FUNCTIONAL DISORDERS OF).

GASTRIC AND DUODENAL ULCERATION (Gastric and Duodenal Ulcer, Simple or Peptic Ulcer).—From the lower end of the oesophagus to the duodenum, at the position of the ampulla of Vater, the gastro-duodenal tract is subjected to the action of the gastric juice, and ulceration may occur in any part of this area.

The ulcer is caused by a necrosis of the mucous membrane which may extend more or less completely through the deeper layers of the wall. It is usually round or oval, with sharply defined edges, and may be small or large, and either acute, with a relatively short duration of symptoms, or chronic, with a long history and corresponding extensive ulcerative changes.

Etiology.—Dealing first with incidence, peptic ulcer is relatively common, and is often present in cases regarded as simple dyspepsia due to hyperchlorhydria. Out of 50,000 autopsies recorded, 4.4 per cent. showed gastric or duodenal ulceration. Surgical and post-mortem experience indicates that the peptic ulcer is found much more often in the duodenum than in the stomach.

Statistics of incidence are, however, of somewhat doubtful value, since those based on clinical observation alone must include cases in which the diagnosis is incorrect, accurate diagnosis on clinical grounds alone often being impossible. On the other hand, it must be remembered that a gastro-duodenal ulcer may heal completely and leave no visible scar; cases of this kind would be excluded from surgical and post-mortem statistics.

Sex.—Moynihan states that gastric ulcer is twice as common in males as in females, and the surgical statistics of others support this view. On the other hand, statistics based on clinical observation give exactly opposite re-

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sults. Post-mortem statistics demonstrate that the incidence in the two sexes is approximately equal.

Age.—Autopsies indicate that in both sexes the disease usually appears between the ages of 20 and 50, the age-period 20 to 30 showing the maximum incidence; but clinical observations favour the view that in females the maximum frequency is between 15 and 30, and in males between 40 and 50.

Heredity seems to play a part in some cases. For example, at St. Mary's Hospital I have met with gastric ulcer in three generations—grandfather, father, and son—in all of whom gastro-enterostomy was performed and an ulcer found at operation.

Trauma and occupation.—Long-continued pressure in the epigastrium may be followed by gastric ulcer. Occupation-compression, such as occurs in shoemakers, tailors, and clerks (from the pressure of the desk), has been recorded as a cause, and the pressure from ill-fitting corsets has also been cited as a factor. A blow in the epigastrium may cause a local injury to the stomach which is followed by the development of a true peptic ulcer in the damaged area, the action of the hydrochloric acid of the gastric juice playing an important part in the subsequent changes. Usually, however, local injuries to the stomach from external violence, provided that rupture has not taken place, heal up without the development of ulcer.

Dietetic errors.—The peptic ulcer has not been shown to be caused by any special article of diet; on the other hand, irregularity of meals, and particularly the habit of going for long periods without food, appears in many cases to play an important rôle not only in the causation of ulcer but also in increasing the symptoms due to an extension of the ulcerating process.

Oral sepsis is undoubtedly a most important factor. Pyorrhœa and oral sepsis are extremely common in gastric and duodenal ulcer, and the infective organisms are unquestionably prominent in the causation. Probably any septic condition of the mouth or naso-pharynx has a predisposing influence, the infecting organisms being carried by the blood-stream to the stomach, or being swallowed and brought into direct contact with it. Recent observations have shown that apical dental abscesses are very frequently the cause of gastric and duodenal ulcer, and of recurrence after treatment. The work of Bolton has shown that in animals the

introduction of septic material into the stomach is followed by the formation of ulcer, and the introduction of dysentery bacilli into the stomachs of guinea-pigs has also produced typical perforating ulcers.

Infections.—Infections of various kinds may be followed by the development of gastric or duodenal ulcer. Of this the well-known occurrence of duodenal ulcer after large superficial burns is an example, the organisms absorbed from the infected area being responsible. General infections such as pyæmia are sometimes a cause of gastric ulcer, and it has also been described as a complication of bacillary dysentery.

Tuberculous ulceration of the stomach is a rare lesion in tuberculosis, and, similarly, syphilitic ulceration very rarely occurs in syphilis. In these cases the ulcer is quite different from the peptic ulcer under consideration here.

Associated diseases.—Anæmia and chlorosis—in both which conditions hyperchlorhydria is common—are important predisposing causes in women. In peptic ulcer in men anæmia is a common sequel rather than a cause. Chronic colitis, especially mucous colitis, is often associated with hyperchlorhydria, and gastric ulcer not infrequently supervenes. Previous gastric disorder—e.g. gastritis—has been said to predispose to ulcer, but the evidence is inconclusive. Arterio-sclerosis and endocarditis have been cited as predisposing causes; the association, however, is unusual.

Pathology.—The pathogenesis of peptic ulcer has given rise to many theories and explanations, and our knowledge of the subject is by no means complete.

It is certain that a local necrosis of a small area of the gastric mucosa occurs, but whether this is produced by a local thrombosis of vessels, or by small emboli, or by the local action of septic organisms in the stomach contents, is not proved; probably one or other of these factors may play a part in different cases.

The action of the hydrochloric acid and digestive ferments on the area of necrosis causes its solution, and an ulcer is thereby produced.

Healing is prevented by the corrosive action of the gastric juice, which may enlarge the area of ulceration. The hydrochloric acid of the gastric juice plays an important but by no means constant part in the production of ulcer. Statistics show that hyperchlorhydria is present in 35–75 per cent. of cases of ulcer, and that

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in only 9 per cent. is the acidity below the normal. Hyperchlorhydria may, of course, be present without an ulcer resulting. When an ulcer is present, healing is not likely to take place unless the hydrochloric acid of the gastric juice is more or less completely neutralized by appropriate alkaline remedies.

Hæmorrhagic erosions and submucous hæmorrhages, which occur not infrequently in appendicitis, in chronic disease of the heart, liver, or kidneys, and in arterio-sclerosis, etc., do not lead, as a rule, to the formation of a true peptic ulcer; it has been claimed, however, that sometimes an ulcer develops on the site of an erosion. This cannot be regarded as proved, and must, at any rate, be extremely rare.

Pathological anatomy.—The distinction between gastric and duodenal ulcer depends upon their relation to the pylorus. On the operating-table the pyloric vein is the best guide.

Ninety per cent. of gastric ulcers are found at the pyloric end of the stomach; and the great majority of duodenal ulcers occur in its first portion, over 50 per cent. of them being within $\frac{1}{2}$ in. of the pylorus and 20 per cent. involving the pyloric ring.

With regard to position in the stomach, post-mortem statistics of 793 cases (Welch) were as follows:—

	<i>Per cent.</i>
Lesser curvature	36
Posterior wall	30
Pylorus	12
Anterior wall	9
Cardia	6
Fundus	4
Greater curvature	3

The ulcers are usually single, but in about 8 per cent. of cases there are more than one.

An acute ulcer is usually small, round, or oval in shape and punched-out in appearance. It is conical, the apex being towards the peritoneal surface of the stomach-wall. The chronic ulcer is of larger size, and its margins are shelved and indurated, and sinuous in outline. Its floor is generally formed either by the submucous or muscular layer of the stomach, but in some cases may be formed by a pancreas which has become adherent to its base. Healing is accompanied by cicatrization, and when the ulcer involves the pylorus the consequent narrowing may cause pyloric obstruction and great dilatation of the stomach. When a large ulcer extends around the stomach

from the lesser curvature in an annular manner its cicatrization may produce an hour-glass contraction.

The inflammatory process may spread through the stomach-wall without causing actual perforation, and perigastric or periduodenal adhesions may result and provoke an additional group of symptoms; in some cases a tumour may be felt in the pyloric region.

Symptomatology.—A gastric or duodenal ulcer may exist for a long period without giving rise to any noticeable symptoms until hæmorrhage or perforation supervenes, so that both have often been found at post-mortem examination when their existence was quite unsuspected during life. Usually, however, definite symptoms are present and show a relationship to food, being increased or diminished by its consumption. There is generally a marked regularity in the occurrence of symptoms, which come on at a definite period after food.

In cases of long standing there is a history of attacks of some weeks' or months' duration, with the intermission of varying periods of good health, occurring with approximate regularity.

Pain is the most constant and important symptom. It usually comes on after the taking of food, and the nearer the ulcer is to the cardia the earlier will be its onset. In gastric ulcer it may be felt immediately or very shortly after ingestion. When it is delayed from one to two hours the ulcer is probably a prepyloric one. Should it be delayed for two hours or more the ulcer is almost certainly duodenal; it then lasts until the taking of food, which usually gives relief for a definite period.

When pyloric stenosis or perigastric inflammation or, possibly, subphrenic abscess is present, the pain becomes more continuous and the regularity of its onset in relation to food becomes lost. This also happens when the ulcer has eroded the stomach-wall and become adherent to the pancreas or adjacent organs. The pain has a definite relation to the kind of food taken; for example, tough meat, salads, pastry, and indigestible articles increase both its severity and its duration. Soft bland foods taken at frequent intervals, on the other hand, minimize the pain, and indeed may lead to its disappearance for a few days.

In character the pain is deep and gnawing, or boring, or like a dull ache; when perigastritis or periduodenitis is present it is more diffuse

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and lancinating. It is usually referred to the epigastric region; in gastric ulcer it is commonly felt just below the xiphisternum, while in duodenal ulcer it is situated midway between xiphisternum and umbilicus. In gastric ulcer it may be referred to the left subcostal border in the epigastric region; in duodenal ulcer, to the right subcostal border. In some cases the pain is also referred to the back, midway between the angles of the scapulæ: in gastric ulcer somewhat to the left, and in duodenal ulcer a little to the right, of the mid-line of the back.

Besides the above symptoms, local spasm set up by the irritation of an ulcer may give rise to sensations of pressure, aching, and fullness of shorter duration. When there is pyloric obstruction, sensations of fullness and of pressure and acid eructations are usually complained of.

Vomiting is often absent or inconspicuous. In gastric ulcer it sometimes comes on shortly after taking food, and is caused by the local discomfort set up, the character of the food having an important influence. When the ulcer is near the pylorus, owing to local spasm or, possibly, actual stenosis the vomiting is obstructive in type and usually occurs in the evening with more or less regularity, a large quantity (two or three pints or more) of acid liquid being brought up. Vomiting directly after food is unusual in gastric ulcer. In this condition vomiting is seldom frequent, and a definite interval elapses which may be longer or shorter according to the position of the ulcer. In perigastritis the vomiting may be frequent and irregular.

Hæmatemesis is not an early symptom, but usually occurs at some period in the course of the disease unless special treatment is adopted. If the hæmorrhage is profuse the vomited blood is red or dark-red in colour and the vomit very acid. When the blood has been in the stomach for some time its hæmoglobin becomes converted by the hydrochloric acid into hæmatin, and the vomit is acid, and somewhat dark in colour—the so-called "coffee-grounds vomit."

It must be remembered that a copious hæmatemesis is a frequent complication of cirrhosis of the liver, gastric erosions, splenic anæmia, and certain toxic conditions; but in these cases the vomit is seldom very acid and has not the appearance of coffee-grounds.

The hæmorrhage of gastric ulcer is due to erosion of a vessel, and recurrences are common,

especially if food by the mouth be not withheld for a few days.

The symptoms due to hæmorrhage are pallor, faintness, dizziness, and amaurosis; collapse follows if the bleeding is severe. In gastric ulcer these symptoms are followed by hæmatemesis, while in duodenal ulcer vomiting may be absent, but much altered blood will be passed in the form of tarry stools (*melæna*). The hæmorrhage is rarely so severe as to be immediately fatal; and it can almost always be controlled by rest and efficient treatment. If occult hæmorrhage is suspected, the patient should be kept on a diet free from meat and blood-containing foods, and the fæces tested daily for blood. The benzidine test is one of the most delicate and convenient. About 10 gr. of benzidine are dissolved in 2 c.c. of glacial acetic acid, and this solution is mixed with double the volume of hydrogen peroxide (B.P.). Some of the fæces are made into an emulsion with water, and to this in a white dish is added the benzidine peroxide solution. A marked blue colour indicates the presence of blood.

Anæmia of a secondary type, loss of weight, and general malaise are common symptoms of chronic ulcer, and result from malnutrition caused by the dyspepsia, and possibly also from actual loss of blood.

Erosion of blood-vessels is a common complication of both gastric and duodenal ulcer, and gives rise to serious hæmorrhage.

Tenderness is usually elicited on careful palpation; it is often localized to a spot in the epigastric region, and is accompanied by definite resistance. When resistance and tenderness are over the right rectus muscle, obvious dilatation of the stomach is present, and may be demonstrated by percussion or auscultation-percussion, while if the tenderness and resistance are situated over the left rectus muscle dilatation of the stomach is usually absent; this is explained by the fact that in the former condition the ulcer is near the pylorus and gives rise to spasm of the constrictor muscle.

A definite *tumour* or thickening can sometimes be felt in the epigastric region, and may be due to the perigastric inflammatory changes set up by the ulcer, or to adhesions between the stomach and neighbouring organs such as the pancreas; in these circumstances a definite mass resembling a new growth may be felt. Occasionally a large chronic ulcer may have its edges and base so thickened that it becomes palpable.

GASTRIC AND DUODENAL ULCERATION

Constipation is often present both in gastric and in duodenal ulcer. The *temperature* is normal or subnormal, except when inflammatory changes follow a partial or complete perforation; in such cases pyrexia is caused by the resulting local or general peritonitis.

When *perforation* occurs gradually and is localized by adhesions the symptoms are indefinite. With a sudden perforation into the general peritoneal cavity, collapse, faintness, and abdominal pain result, the temperature is subnormal, and pronounced resistance and abdominal tenderness are present. In a few hours, should the patient survive the shock of the perforation, the temperature rises and the usual symptoms of general peritonitis supervene, which, in the case of a duodenal ulcer, are most marked on the right side of the abdomen. When perforation takes place into the lesser peritoneal cavity, or when there is localization by adhesions—e.g. in subphrenic abscess—there are, in addition to the usual features of a localized peritonitis, the physical signs of an air-containing cavity in which fluid is present. Usually a bell-note can be obtained over the affected part, sometimes splashing can be heard on auscultation, and the area of dullness varies with the position of the patient.

Hour-glass contraction of the stomach usually gives rise to pain soon after food, and frequent vomiting. On palpation much resistance and thickening are felt in the epigastric region. The condition is one which causes progressive emaciation, and there are no periods of freedom from symptoms. The diagnosis is made by X-ray examination.

Carcinoma developing on ulcer.—A chronic ulcer, especially if it be situated near the pylorus, may become carcinomatous. It has been estimated that in from 5 to 10 per cent. of cases ulcer is followed by growth, but the statistics of different observers vary greatly. Some American surgeons maintain that the development of carcinoma can be detected in over 50 per cent. of resected ulcers. In Great Britain physicians usually regard the sequence as rare, while surgeons hold that it is common. A chronic gastric ulcer may show great thickening of its edges and base from inflammatory changes, and may be mistaken for a new growth; but microscopic examination at once differentiates the two conditions.

Jejunal peptic ulcer occurs, though very rarely, after the operation of gastro-jejunos-

tomy. In many of these cases the ulcer has involved both stomach and duodenum.

Diagnosis.—Ulcer has to be carefully differentiated from other gastric conditions, such as chronic gastritis, new growths, and motor, sensory, or secretory neuroses of the stomach.

Other causes of epigastric pain have to be carefully considered; such are biliary colic, the gastric crises of tabes dorsalis, and the pain associated with spinal disease, e.g. caries.

Other causes of vomiting must be borne in mind; these include infective conditions (e.g. tuberculosis, in which vomiting may be a prominent symptom), auto-intoxication (such as uræmia, acidosis, and liver atrophy), and chemical poisons (such as arsenic) and other irritant or toxic substances.

Other causes of hæmatemesis, such as the hæmorrhagic blood conditions, cardiac, vascular, or renal disease, splenic anæmia, cirrhosis of liver, and gastric erosions, must be distinguished.

The diagnosis between these various diseases and ulcer is made to a certain extent from a careful consideration of the clinical symptoms, but an exact diagnosis is impossible on these grounds alone. The most valuable and exact aids to diagnosis are furnished by (1) the test-meal analysis, (2) X-ray examination.

(1) The details of the *test-meal examination* are given in the article on GASTRIC CONTENTS, EXAMINATION OF. In gastric and duodenal ulcer, free hydrochloric acid is usually present. The active hydrochloric acid is increased and is usually above 0.2 per cent. Mucin and lactic acid are generally absent. The ferment activity is, as a rule, much above the normal.

The result serves to differentiate ulcer from growth of the stomach and chronic gastritis, and is also of value in differentiating other forms of vomiting such as those due to infections and toxæmias.

(2) The *X-ray examination* is made after a meal containing barium sulphate or bismuth carbonate, and by its means delay in the emptying of the stomach may be demonstrated—an evidence of pyloric obstruction.

Excessive peristalsis and rapid emptying of the stomach are seen in certain types of duodenal ulcer.

In gastric ulcer a sustained contraction of the circular muscle-fibres is usually provoked, so that at the site of the ulcer the stomach

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is partially bisected by the constricting band of muscle. When the ulcer is situated in the lesser curvature a distinct incisura is shown on the greater curvature opposite. Direct evidence of an ulcer is often obtained by X-ray examination, the crater of the ulcer becoming filled with the opaque meal, which often appears as an outlying excrescence.

An X-ray examination should be made in every case in which gastric or duodenal ulcer is suspected; it is a most valuable method of diagnosis. (See PLATE 46, Fig. 5, and PLATE 47, Fig. 1, Vol. III, facing pp. 557, 562.)

Prognosis.—When the history is of short duration there is a reasonable prospect of cure if the patient submits to a long rest and careful medical treatment.

When perforation occurs, especially if general peritonitis is set up, the prognosis is very grave. Should the patient be operated upon within twelve hours of the rupture the mortality is 25 per cent.; but should twenty-four hours elapse there is very little chance of recovery from operation, the mortality being given as 95 per cent.

Hæmatemesis is a grave complication, but with very careful medical treatment it is rarely directly fatal.

Cases which are not likely to be cured in a reasonable time by medical measures are usually treated surgically, and the results obtained in recent years have been very encouraging.

The mortality from gastro-enterostomy in cases of peptic ulcer (excluding perforation) is very low, in some statistics under 1 per cent., and the prospects of cure, or at any rate of complete relief from symptoms, are good.

Treatment.—When perforation has occurred the patient should be operated upon with the least possible delay.

When, in cases of pyloric ulcer, almost complete obstruction occurs, nearly all the food taken by the mouth being vomited up at some time during the day, it is unwise to attempt any special medical treatment beyond that of rendering the patient fit to withstand the operation of gastro-jejunostomy as soon as possible; thus, antiseptic treatment of the mouth, gastric lavage, and the free administration of normal saline by the bowel should be adopted for a few days as preparatory treatment.

Apart from these urgent complications, it is advisable to submit all cases of peptic ulcer

to a thorough course of medical treatment, and there is reason to believe that in a considerable percentage of cases actual healing of the ulcer occurs and a good recovery is made.

Medical treatment.—Should the condition of the patient admit, the first consideration is to remove any source of oral sepsis. Carious stumps should be extracted and an X-ray examination made of the teeth; those showing marked signs of septic changes round them should be removed. Suitable antiseptic treatment of the remaining teeth and the use of an autogenous parodontal vaccine are advisable. Absolute rest in bed should be enforced. Local applications to the epigastrium in the form of poultices, antiphlogistine, ice, etc., may be used if the pain is severe. When there has been frequent vomiting or hæmatemesis, it is advisable to withhold food by the mouth for four or five days, and to give rectal feeding every six hours. Suitable *nutrient enemata* are:

- Ry Somatose 3ss.
Glucose 3vi.
Normal saline 3x.
- Ry Glucose 3vi.
Yolks of two eggs.
Salt gr. viii.
Peptonized milk 3x.

Normal saline, 15 or 20 oz., given every eight hours, probably answers equally well. A few teaspoonfuls of hot water may be sipped if thirst is severe. After the period of rectal feeding, the diet advised by Lenhartz may be given: it consists of 8 oz. of milk mixed with one egg for the first twenty-four hours; then 4 oz. of milk and one egg are added daily until two pints of milk are given. Afterwards a little scraped raw meat can be given and, later, boiled rice, pounded fish or chicken, the number of eggs being reduced. It is advantageous to use citrated milk in this dietary. In place of the Lenhartz diet, citrated or peptonized milk to which has been added sodium bicarbonate 1 dr. to the pint may be given; this may be supplemented by malted milk, plasmon, or beaten-up eggs.

In cases of chronic ulceration in which the symptoms do not call for a course of treatment so rigorous as the above, the patient should be put on a liquid diet. Citrated milk in which eggs are beaten up, Benger's food, or custard may be given. About 5 oz. of liquid should be taken every two hours, and, if tolerated, milk puddings and pounded fish may then be added.

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The following mixture should be ordered :—

Ry Bism. carb. gr. xx.
Mag. carb. gr. x.
Sod. bicarb. gr. lx.
Sp. chlorof. ℥x.
Glycer. acid. carbol. ℥x.
Aq. menth. pip. ad ʒi.
T.d.s.

Surgical treatment is indicated (1) for perforation, (2) when definite pyloric obstruction exists, (3) in cases in which there have been repeated attacks of hæmatemesis, (4) in cases of chronic ulcer in which medical treatment has failed to give permanent relief.

In hæmatemesis it is not advisable to operate until the patient has recovered sufficiently from the last hæmorrhage to withstand the shock of the operation. If the hæmorrhage has been very severe it is wise to give a liberal transfusion of blood before operating, since this not only diminishes the risk from the operation but permits its being done at an earlier date.

The operation usually performed for peptic ulcer is that of *gastro-jejunostomy*, which is generally followed by disappearance of symptoms and by healing of the ulcer. It is usually combined with local surgical treatment of the ulcer, such as cauterization, or excision by a *partial gastrectomy*, since in this way the possible development of carcinoma on the site of the ulcer is prevented.

The surgical treatment of gastric and duodenal ulcer has given brilliant results, and the mortality from the operation is very low.

It cannot be urged too strongly that operative treatment should not be adopted until the existence of an ulcer has been demonstrated by the diagnostic methods described, for it is in cases in which an actual ulcer is found that the results are so beneficial.

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GASTRIC CATARRH (see GASTRITIS).

GASTRIC CONTENTS, EXAMINATION OF.—A careful examination of the gastric contents after a test meal is required in all cases presenting persistent gastric symptoms which do not yield to medical treatment, since by this means the diagnosis can be placed on a surer basis and the correct course of treatment more clearly indicated. The diagnosis, however, should not rest on the result of the test-meal examination alone. In every case, at the outset, the most careful investigation should be made into the clinical history,

symptoms, and physical signs, and this should be supplemented by an examination of the gastric contents after a test meal, and usually also by a series of X-ray examinations of the gastro-intestinal tract.

The test-meal result will indicate which of the alternative clinical diagnoses is the correct one, and the X-ray examination will give further information, from the mechanical changes in the size, shape, and motor functions of the stomach, as to the exact pathological condition present.

It cannot be too strongly urged that whenever an operation is to be performed for symptoms referable to the stomach a preliminary examination of the gastric contents on chemical lines should, if possible, be made, since in many cases this will not only guide the surgeon to the probable diagnosis, but will often suggest to him the best course of procedure after the abdomen is opened, and whether a gastro-enterostomy is likely to be of benefit. In acute perforative lesions of the stomach and duodenum, however, previous investigations are quite out of the question, since immediate operation is imperative.

The composition of the gastric contents after a test meal varies according to the pathological condition of the stomach, and a particular disease is associated with a gastric content of a definite type.

For example, in a case in which there have been persistent gastric symptoms for many months, with loss of weight and anæmia, and a localized resistance or possibly a tumour is felt in the epigastric region, the gastric analysis will usually enable a differential diagnosis to be made between new growth and chronic ulcer of the stomach, and furnish valuable information as to prognosis and treatment.

Similarly, in a case presenting chronic dyspeptic symptoms with dilatation of the stomach, the test-meal examination serves to distinguish between chronic gastritis and gastric ulcer and to indicate the line of treatment to be adopted.

1. How the "gastric contents" are obtained.—Analyses of vomit do not give satisfactory results as to the condition of the gastric contents, since much admixed saliva is present, and vomiting is likely to occur at a time when much fermentation has been taking place, so that the percentage of organic acids and of other products of fermentation is likely to be abnormally high and misleading.

When a case comes under treatment it is

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well for the patient to rest in bed for a day or two on a restricted and light diet, no medicines being given, so that any temporary dilatation of the stomach may be reduced. Usually it is unnecessary for the stomach to be washed out before giving the test meal, but in cases where great dilatation is present it is advisable to wash it out with water and completely empty it on the day before the test meal is given.

The most suitable and palatable test meal is $1\frac{1}{2}$ pints of weak tea with a little milk and sugar to taste and a round of thin buttered toast.

In place of this may be given a gruel test meal, made by mixing three tablespoonfuls of oatmeal with 3 pints of water, the mixture being slowly boiled down to $1\frac{1}{2}$ pints and strained through muslin. The gruel should be taken warm, and salt added to taste if desired.

It is important that a test meal should not contain substances which will interfere with the subsequent analysis, and on this account meat, fish, egg, or much milk is to be avoided.

For an ordinary examination the tea-and-toast meal is the most convenient. For a fractional test meal, gruel is preferable.

The test meal is best given in the morning before any other food is taken, and withdrawn after an interval of an hour.

It is most important that there should be no dilution with water in the withdrawal of the gastric contents, since this would vitiate the analytical results.

The patient should sit upright in bed or in a chair, the head not being thrown back. The mouth should be widely opened and a soft stomach-tube which has been lubricated with oil or a little glycerin is passed down the pharynx and oesophagus to the stomach. The tube is held in the right hand of the operator and is guided along the posterior wall of the pharynx by his left forefinger. Attached to the soft stomach-tube should be about 3 ft. of indiarubber tubing, from the end of which the air is withdrawn by suction with an ordinary glass syringe. In this way siphon action is started and the gastric contents will flow gently out on removal of the syringe.

The stomach should be emptied as far as possible, and the quantity withdrawn measured and reserved for analysis. Usually 4-6 oz. are obtained, but in cases of pyloric obstruction the quantity is considerably greater.

II. General examination of the gastric contents.—A portion is taken and allowed to settle, the deposit being examined microscopically. In addition to the starch granules present in the test meal, bacteria such as sarcinae, long bacilli, or yeast cells may be found. Red blood-corpuscles should be looked for. Epithelial cells are commonly present, and are especially numerous when gastric carcinoma is present.

The remainder of the gastric contents is examined as to consistence, odour, colour, etc.

In gastritis or carcinoma the contents are viscid from the large quantity of mucus present. A sour acid smell indicates the presence of organic acids and fermentative changes.

The gastric contents are then filtered, and the filtrate used for the subsequent tests.

In the case of the tea test meal the filtrate is straw-coloured; with the gruel meal it is colourless. Should bile or blood be present the colour will be affected.

Bile may be tested for by the iodine test, and blood by the guaiacum and ozonic ether test or by the spectroscope.

III. Special chemical examination.

1. Free hydrochloric acid.—The best qualitative test for this is *Günzberg's*. About 2 gr. of vanillin and 4 gr. of phloroglucin are placed in a porcelain evaporating dish and 1 c.c. of alcohol is added to dissolve them; about 2 c.c. of the filtered gastric contents are added, and the dish is placed on the water-bath till its contents are nearly dry. A brilliant scarlet-red colour is produced if free hydrochloric acid is present.

Di-methyl-amido-azo-benzene in 1-per-cent. solution in alcohol may be used (*Töpfer's test*). A drop of this solution added to a few drops of the gastric contents gives a pink-red colour if free HCl is present.

The test may be used quantitatively. Thus, if to 10 c.c. of the filtered gastric contents are added two or three drops of the reagent, a pink-red colour is produced. If now decinormal caustic soda solution is run in from a burette until the red colour just disappears, the quantity added is a measure of the free hydrochloric acid present.

Where much organic acid is present a red coloration may be given by *Töpfer's test*, so that, though commonly used in the determination of free hydrochloric acid, the reaction is not one of great accuracy.

Congo-red test paper is turned blue by free

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hydrochloric acid and is a convenient test. The reaction is, however, given where much organic acid is present, and is therefore not very reliable.

Too much importance should not be attached to the presence or absence of free hydrochloric acid in the gastric contents. Protein in the form of albumin, albumose, or peptone is always present in the gastric contents, and combines with the free hydrochloric acid secreted by the stomach, forming protein-combined hydrochloric acid; also some of the free HCl may be combined with nitrogenous organic bases which are produced in digestion.

The hydrochloric acid which is free, together with that which in the process of digestion has combined with protein and nitrogenous organic bases, is the physiologically active hydrochloric acid and may conveniently be termed "active hydrochloric acid." This active hydrochloric acid is by far the most important estimation in the test-meal analysis, and it is essential that an accurate method of determining it should be adopted.

2. Determination of the active hydrochloric acid.—In the gastric contents hydrochloric acid is present in three forms:

- Active HCl { (1) Free hydrochloric acid.
(2) Hydrochloric acid combined with protein and nitrogenous organic bases.
(3) Hydrochloric acid combined with inorganic bases, e.g. sodium chloride.

It is the accurate estimation of (1) and (2) which, as I have said, is all-important.

The usual methods of titration with caustic soda solution, using different indicators, are in my experience unreliable and of insufficient accuracy.

The following method is very accurate and easy of performance: 10 c.c. of the filtered gastric contents are placed in each of two porcelain evaporating dishes, A and B. To A is added some pure sodium carbonate in excess. The two dishes are placed on the water-bath and the contents evaporated to dryness. The dishes are now gently ignited until the contents are completely charred. When they are cold, about 60 c.c. of water are added to each dish, also 10 c.c. of pure nitric acid and 5 c.c. of a 5-per-cent. solution of iron alum. The contents are well stirred, and 30 c.c. of decinormal silver nitrate solution are added to each. A decinormal solution of ammonium sulphocyanide solution is run from a burette

into each dish until a permanent pink colour remains.

The difference between the amount of silver nitrate solution taken (30 c.c.) and the number of cubic centimetres of ammonium sulphocyanide solution used corresponds to the quantity of hydrochloric acid present, expressed in terms of cubic centimetres of a decinormal solution.

The amount of hydrochloric acid found in dish A corresponds to the free hydrochloric acid (1) + the hydrochloric acid combined with protein and nitrogenous organic bases (2) + the hydrochloric acid combined with inorganic bases (3), since the addition of sodium carbonate before ignition fixes all the hydrochloric acid.

In dish B ignition drives off the free HCl (1) and the HCl combined with protein and nitrogenous organic bases (2), so that the hydrochloric acid actually found is that combined with inorganic bases (3).

The difference between the results of A and B, therefore, gives the amount of free HCl and HCl combined with protein and nitrogenous organic bases—that is, the *active hydrochloric acid*. If this figure is x c.c., the quantity of active hydrochloric acid in 100 c.c. of gastric contents is

$$x \times 10 \times 0.00365 \text{ gm.},$$

which gives the percentage of active hydrochloric acid present.

The normal percentage of hydrochloric acid is between 0.1 per cent. and 0.2 per cent.; usually it is about 0.15 per cent.

3. Total acidity.—This is determined by taking 10 c.c. of the filtered gastric contents, diluting with about five times the quantity of distilled water, and, after adding a few drops of phenolphthalein solution as indicator, a decinormal solution of barium hydrate is added until a permanent pink colour is produced.

The total acidity is calculated in terms of hydrochloric acid, and is normally between 0.1 and 0.2 per cent.

Before the titration is performed the gastric content should be warmed and a current of air passed through it for two or three minutes to drive off any dissolved carbonic acid which would affect the accuracy of the titration.

$\frac{n}{10}$ barium hydrate standard solution gives more accurate results than $\frac{n}{10}$ sodium hydrate, though the latter is often used.

4. Organic acids.—Of these, lactic acid is the

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chief. It is tested for by adding to the filtered gastric contents some *Uffelmann's reagent* (the purple-coloured liquid obtained by mixing weak solutions of ferric chloride and carbolic acid). A yellow coloration is produced.

The quantity of organic acids present is given by the difference in percentage between the total acidity and the active hydrochloric acid results.

5. **Mucin** is tested for by adding 2-per-cent. solution of acetic acid to the gastric contents, when a white precipitate is produced.

6. **Sulphocyanides** are shown to be present by the red colour resulting when a few drops of ferric chloride solution are added. The colour is not affected by hydrochloric acid, but is destroyed by mercuric chloride solution.

Sulphocyanides come from the saliva and, if present in distinct amount, discount the presence of mucin which may come from the same source.

7. **Ferment activity.**—This is a most valuable guide, and should always form part of the examination of the gastric contents. The most convenient and accurate method of determining this is by estimating the rennin present. It has been found that this gives an accurate measure of the pepsin and pepsinogen present. Five cubic centimetres of fresh unboiled milk are placed in each of a series of 10 test-tubes, $\frac{3}{8}$ in. in diameter. The tubes are placed in a water-bath at 40° C., and quantities of the filtered gastric contents, 0.01 c.c., 0.05 c.c., 0.1 c.c., 0.15 c.c., 0.2 c.c., 0.25 c.c., 0.3 c.c., 0.4 c.c., 0.5 c.c., 0.6 c.c., are added respectively to each and mixed.

In half an hour the tubes are taken out of the bath and inverted. In those in which coagulation has taken place the contents remain in the tubes, in the others they flow out.

In normal gastric contents 0.2 c.c. is the minimum amount which causes coagulation.

If x c.c. are required, the ferment activity is represented by $\frac{0.2}{x}$ times the normal.

Conclusions to be drawn. 1. *Gastric and duodenal ulcer.*—Free HCl is usually present. The active HCl is increased and is commonly above 0.2 per cent. Mucin and lactic acid are generally absent. The ferment activity is, as a rule, much above the normal.

2. *Hyperchlorhydria.*—The results are similar to those of gastric and duodenal ulcer, so that the clinical and X-ray examinations are necessary for differential diagnosis.

3. *Gastric carcinoma.*—Free HCl is almost always absent, and the active HCl is much below normal, usually less than 0.1 per cent. Lactic acid and mucin are commonly present. The ferment activity is much below normal. Lactic acid is generally present, and in cases of pyloric carcinoma the organic acids may be high so that the total acidity is above normal, but in these cases the active HCl is usually much diminished.

4. *Chronic gastritis* is associated with absence of free HCl, diminution of active HCl, and low ferment activity. Mucin and lactic acid are often present. The results resemble those of gastric carcinoma, and the clinical and X-ray examinations serve to differentiate the conditions.

5. *Chlorosis* is usually associated with hyperchlorhydria.

6. *Gastralgia and neurasthenia* with gastric symptoms generally give normal results after test-meal examination, but hyperchlorhydria may be present.

7. In *atonic dilatation of the stomach* the results are usually rather below the normal as regards active HCl and ferment activity.

8. *Mucous colitis* is frequently associated with an increase in active HCl and ferment activity.

9. *Cholelithiasis* is often associated with chronic gastritis, and the test-meal results correspond to that condition. Sometimes, however, hyperchlorhydria occurs.

10. *Cirrhosis of the liver* usually gives results corresponding to those of chronic gastritis.

11. *Pernicious anaemia* and *achylia gastrica* show absence of free HCl and a great diminution of active HCl. The ferment activity is almost entirely absent.

12. In cases where there is a communication between the stomach and transverse colon the primary lesion present is generally a carcinoma of the stomach or the colon. In the former case the test-meal analysis will show the characters of gastric carcinoma, in the latter it will be approximately normal. In both, however, the test for stercobilin will be positive. If to the filtered gastric contents a solution of zinc chloride in ammonia and alcohol is added, a well-marked fluorescent liquid is obtained showing the presence of stercobilin.

The fractional test-meal.—During the last few years investigations of the gastric contents after a gruel test-meal of 1 pint

GASTRITIS, ACUTE

have been made by withdrawing small quantities every quarter of an hour and analysing them.

A soft rubber tube of diameter equal to that of No. 6 or No. 8 soft catheter is used, and attached to it is a small oval metal bulb containing numerous perforations. The bulb and portion of the tube are swallowed so that the bulb enters the stomach cavity.

Small quantities of the gastric contents are withdrawn at the required intervals by means of a record syringe.

Analyses are made of the specimens obtained and a curve of the results is plotted. The method gives a sharp determination of the time of emptying of the stomach, which is indicated by the disappearance of starch from the gastric contents.

At present it would be premature to draw any conclusions as to the advantages in diagnosis gained by this method of examination, but there is no doubt that it will disclose much information concerning the normal processes of digestion. It is probable also that it will yield further knowledge of the composition of the gastric contents in pathological conditions of the stomach.

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GASTRIC CRISES OF TABES (*see* STOMACH, FUNCTIONAL DISORDERS OF).

GASTRIC DILATATION (*see* STOMACH, FUNCTIONAL DISORDERS OF).

GASTRIC JUICE, ABNORMAL SECRETION OF (*see* STOMACH, FUNCTIONAL DISORDERS OF).

GASTRIC LAVAGE (*see* GASTRITIS, ACUTE; GASTRITIS, CHRONIC).

GASTRIC MOTILITY, DISORDERS OF (*see* STOMACH, FUNCTIONAL DISORDERS OF).

GASTRITIS, ACUTE (*syn.* Acute Gastric Catarrh).—An acute inflammation of the gastric mucosa accompanied by symptoms of local and perhaps also of constitutional disturbance. It occurs at all ages.

Acute gastritis may be primary, being caused by some error in diet or some physical condition such as chill, fatigue, debility, or nervous exhaustion. But the affection is very commonly *secondary* in origin, being a symptom and part of the symptom-complex of some more important disease. Thus, it may be due to—

- (1) Irritant poisoning, such as that arising from the mineral acids, caustic alkalis, arsenic, and phosphorus.
- (2) The toxæmia of some infectious disease, such as influenza, smallpox, or scarlet fever; in these it appears as an early symptom.
- (3) Bacterial food-poisoning—so-called "ptomaine poisoning," in which a gastritis and usually also an enterocolitis are set up by the contamination of foods by certain pathogenic bacteria.
- (4) Some other abdominal disease, for example gastric ulcer, appendicitis, renal or biliary colic, cirrhosis or some other disease of the liver.
- (5) Auto-intoxications, such as uræmia, or the vomiting associated with various types of acid intoxication, such as recurrent vomiting in children.
- (6) Some disease of the nervous system, e.g. the gastritis associated with the gastric crises of tabes dorsalis.

The conditions to which acute gastritis is secondary are considered in the articles on those subjects. Here we are concerned with primary acute gastritis only.

It is important that a thorough examination of every system should be made in all cases; and primary acute gastritis should never be diagnosed until all possible causes of secondary gastritis have been excluded.

Etiology.—Primary acute gastritis is a common affection of young children, some food which the stomach cannot digest acting as an irritant and causing acute gastric symptoms.

In adults food is a common cause: thus a large indigestible meal may cause gastritis in a person of normal digestion; or in a patient with a delicate digestion, ordinary articles of diet may have an irritant effect on the stomach—for this reason convalescents from acute diseases require careful and graduated dieting.

Foodstuffs vary much in their digestibility, and indigestible articles, such as pork, salmon, lobster, mackerel, etc., may give rise to acute gastric catarrh in healthy persons.

General diseases, such as malaria, gout, and rheumatism, predispose to attacks.

Alcohol, tobacco, and narcotic drugs are common causes. Attention has been called by Dr. John Freeman to the susceptibility of certain individuals to toxic effects as a consequence of the taking of particular foods;

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to these cases the term "toxic idiopathy" has been applied. Thus, in some persons, rabbit, horseflesh, eggs, or even honey may set up symptoms of acute gastritis and constitutional disturbance. These articles must, of course, always be avoided by susceptible individuals.

Symptomatology.—There is usually discomfort with a sense of distension and fullness in the epigastrium, and in some cases there may be acute pain. Anorexia, salivation, nausea, acid eructations, pyrosis, and vomiting commonly occur. The tongue is much furred, and usually dryish and indented by the teeth; this symptom is associated with foul breath. Heartburn and pain round the heart may be present. General prostration and malaise are accompanied often by vertigo, and also by headache, which may be frontal, temporal, or occipital in position, and perhaps of a hammering character. Constipation is usual, except in cases such as food poisoning associated with enteritis, when diarrhoea occurs. In adults there is usually no fever; indeed, it may be taken as an axiom that if gastritis is associated in an adult with fever lasting some days the stomach condition is certainly secondary.

Cases have been described associated with herpes and fever, but in these some general infection is the primary cause and the gastritis is secondary. In children, however, simple gastritis is not uncommonly accompanied by a rise of temperature.

In some cases the sclerotics may be icteric, and definite jaundice may develop later.

The vomit usually contains much mucus and is acid, but free hydrochloric acid, which may be shown by the blue coloration of congo-red paper, is generally absent.

Examination of the abdomen usually shows some general tenderness in the epigastrium, but this is not localized to one spot, and no tumour or local thickening is to be felt on palpation. Percussion or auscultato-percussion may reveal great dilatation in certain cases, and splashing may be obtained. Dilatation is, however, the exception rather than the rule.

Diagnosis.—The nature of the affection is indicated by the acute onset of characteristic symptoms traceable to a definite etiological factor, the progress of the case, and the careful exclusion of all possible causes of secondary gastritis or other diseases which could give rise to the symptoms.

Prognosis.—Primary acute gastritis usually responds readily to treatment, and the symptoms quickly clear up.

Treatment.—Removal of the irritating contents of the stomach is the first consideration. This is usually effected by the vomiting associated with the complaint, and assistance may be given by the drinking of warm water to which bicarbonate of soda (30 gr. to the pint) has been added.

In cases in which there is evidence of dilated stomach, or where the vomiting is very persistent, **gastric lavage** is indicated. This should be applied by the passage of a soft stomach-tube to which rubber tubing (about 3 ft. in length) with funnel is attached. The stomach can then be washed out by siphonage with a warm solution of bicarbonate of soda (30 gr. to the pint), and should afterwards be emptied.

Rest in bed is essential, and food should not be given by the mouth until the symptoms of gastric irritation have subsided. Sips of hot water or the sucking of small pieces of ice may be allowed to allay thirst.

If vomiting persists after these measures, it is well to give the stomach complete rest and to adopt **rectal feeding**. The bowel is emptied by means of a simple enema and washed out with normal saline solution. Then 15 oz. of normal saline containing 2 per cent. of glucose are slowly given per rectum by means of a soft rubber catheter and funnel every four hours during the day, no food being given by the mouth.

Local applications in the epigastric region, such as mustard leaves, mustard poultices, fomentations, or the icebag, are helpful. When food can be taken by the mouth, albumen-water containing 4 per cent. lactose, or normal saline, or citrated milk (2 gr. of citrate of soda to each ounce of milk) diluted with double the quantity of water or Vichy water, may be allowed during the first day or two.

It is important that the urine be tested for acetone. If this is present it is advisable to give bicarbonate of soda in full doses by the mouth, e.g. 30 gr. every two hours in a little soda water; it may also be added (1 dr. to the pint) to the rectal feeds.

Medicinal treatment.—Usually the above treatment suffices, but when the gastric irritation continues the following mixture may be given every three hours:—

Ry Bism. carb. gr. xx.
Mag. carb. gr. x.
Sod. bicarb. gr. xxx.
Acid. hydrocyan. dil. ℥iii.
Mucil. trag. ʒi.
Aq. menth. pip. ad ʒi.

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When there is much pain, 10 min. of liq. morphine mur. may be added to each dose, or a hypodermic of morphine ($\frac{1}{4}$ gr.) given.

If the vomiting is very persistent and does not respond to the foregoing treatment, cerium oxalate (3 gr.) may be added to the bismuth prescription.

It is important that intestinal elimination should be secured by the administration of an appropriate purgative such as calomel (1 or 2 grains) at night, followed by a saline aperient in the morning.

Diet.—When the stomach can retain food the lightest diet should be given at first, such as citrated milk diluted with an equal quantity of barley water or chicken broth; gradually milk puddings, soups, fish, and pounded chicken may afterwards be allowed. When convalescence is established, it is important that the greatest care be observed with regard to diet in order that a recurrence of the illness may be avoided.

In secondary acute gastritis, besides the appropriate treatment of the causal condition, measures such as those here described for primary acute gastritis may be adopted with advantage.

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GASTRITIS, CHRONIC.—A chronic catarrhal inflammation of the mucous membrane of the stomach, resulting in changes in the gastric secretion and motor power with disturbance of digestion.

Chronic gastritis may be *primary*, when the gastric inflammation is the sole cause of the symptoms. It is important to remember, however, that in a large number of cases it is *secondary*. Thus secondary chronic gastritis may be due to—

- (1) Some organic disease of the stomach, e.g. new growth or chronic ulcer.
- (2) Some other abdominal disease, e.g. gall-stones, cholecystitis, chronic appendicitis, liver or pancreatic disease, colitis.
- (3) Conditions of portal congestion causing engorgement of the gastric mucosa, e.g. cirrhosis of the liver, chronic heart and lung diseases.
- (4) Chronic poisoning, such as occurred in aeroplane workers from tetrachlorethane vapour, among munition workers from trinitrotoluene and other toxic substances; also chronic poisoning from arsenic, phosphorus, etc.

(5) Auto-intoxications, such as the gastritis associated with chronic renal disease; diabetes, acid intoxications, etc.

(5) Nervous diseases, e.g. (a) intracranial lesions such as tumour, chronic meningitis, etc., giving rise to headache, optic neuritis, and vomiting; (b) the gastric crises associated with tabes dorsalis, in which the symptoms are often accompanied by some chronic gastritis.

It is important that a thorough examination of every symptom be carefully made in all cases. Primary chronic gastritis should never be diagnosed until all possible causes of the secondary form of the affection have been excluded.

In the present article we need not further consider secondary chronic gastritis; for this the articles on the antecedent conditions should be consulted.

Etiology.—The causes of primary chronic gastritis may be thus classified:

1. **Dietetic.**—The habitual taking of unsuitable foods, such as indigestible articles containing a high proportion of fat, an excess of starchy foods, or the immoderate consumption of tea and coffee, are common causes.

Perhaps the most frequent predisposing cause is irregularity of meals; the patient has gone too long without food, and then perhaps taken starchy foods which are not digested in the stomach. Imperfect mastication due to defective teeth is often at fault. Also, the custom which prevails among those engaged in business of taking hurried meals followed by an immediate resumption of work, gives the stomach no opportunity for digestion. Habitual consumption of meals too large for the stomach to digest is an obvious cause, but in these days of privation is not met with so often as formerly.

The consumption of alcohol is an important cause; persons who habitually exceed are constantly subject to chronic gastritis. The taking of alcohol on an empty stomach and the custom of "having drinks" at all times with acquaintances frequently lead to chronic gastritis, as do excessive smoking or chewing of tobacco, and the habitual taking of opium and other narcotic drugs.

2. **Constitutional causes.**—Gastritis is one of the commonest early symptoms of anæmia, which may be due to physiological conditions such as the chlorosis occurring in young women,

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or may be the commencement of a grave primary anæmia of pernicious type.

3. Among the common causes are **infective conditions** associated with the continued ingestion of some septic or toxic matter. Examples are pyorrhœa and septic conditions of the mouth or naso-pharynx, unhealthy tonsils, adenoids, and suppuration of the maxillary antra or sinuses communicating with the nasal passages.

4. **Neurasthenia** induced by worry and overwork often acts as a predisponent.

Pathology.—Two forms are met with—(1) the productive or hypertrophic, and (2) the atrophic.

(1) The **hypertrophic** form is the commoner. In this the stomach is enlarged, except in the cirrhotic variety, in which there is shrinkage. Much thick greyish mucus, which may be blood-stained, is found adherent to the mucous membrane. The stomach wall is thickened and of a greyish or greyish-red colour, reddish patches being often found in the pyloric portion, due to local congestion of the vessels. Small hæmorrhages may be present, and in old-standing cases there may be some pigmentation due to hæmorrhagic extravasation. Small erosions are not uncommon and may give rise to minute superficial ulcers due to inflammatory hyperplasia of the lymph-follicles, with subsequent degeneration. The existence of these lesions explains the hæmatemesis which occurs in cases of this disease. The mucous membrane of the stomach, by its general thickening, may have in places an extremely wrinkled and mamillated appearance.

(2) **Atrophic form.**—The wall of the stomach is thinned, the mucous membrane being pale, thin, and smooth. There is also pronounced atrophy of the secretory glands, so that only a thin membrane covers the muscularis mucosa and muscular layers, which may be toughened by fibrosis.

Symptoms.—Usually with a lack of appetite there is a sense of oppression and discomfort after meals, which may amount to actual pain, and is referred to the epigastric region, or sometimes to the præcordium, causing the sensation of heartburn. A disagreeable taste in the mouth is often present, and the tongue may be furred and its margin reddened, and indented at the edges by the teeth.

Marked gastric flatulence with eructations of gas is the rule. Nausea with perhaps retching, pyrosis (waterbrash), and vomiting are common. The vomit generally contains much

slimy mucus and food in various stages of digestion. Organic acids, such as butyric and lactic acids, are often found, but free hydrochloric acid is usually absent.

Constipation and intestinal distension and flatulence may be present; diarrhœa is uncommon.

General symptoms.—Headache, lassitude, and depression are often complained of; ocular symptoms such as spots in the field of vision (*muscæ volitantes*) occur; vertigo is not uncommon. Irregularity of the pulse due to extrasystoles may be accompanied by cardiac discomfort and distress, and sometimes there are attacks of præcordial pain (*pseudo-angina*). In patients suffering from myocardial degeneration, or sclerosis of the coronary vessels, chronic gastritis may reflexly cause fatal syncope.

Insomnia and symptoms of neurasthenia are not unusual.

Physical examination usually reveals some discomfort on palpation over the stomach, and dilatation may be made out by percussion or auscultato-percussion. Splashing due to excessive liquid in the stomach may be obtained. No tumour or thickening in the gastric region will be detected, except in cases of secondary chronic gastritis.

Diagnosis.—In mild cases the symptoms usually clear up quickly with appropriate treatment.

When they are of long standing and have resisted treatment it is essential that special investigations should be made to discover the actual fault in the digestive processes. The symptoms may be due to some defect in the secretion or motility of the stomach if the condition is primary in origin. The teeth should be examined carefully for evidence of pyorrhœa, and an X-ray examination is advisable should there be any suspicion of disease here.

The tonsils, maxillary antra, and naso-pharynx should also be examined carefully for any evidence of sepsis.

The **gastric secretion** is best determined by a test-meal examination. A pint of weak tea with some buttered toast is given in the early morning, and an hour later the gastric contents are withdrawn, without dilution, by means of a soft tube and a funnel. The active hydrochloric acid is usually much diminished (the normal percentage being 0.15–0.2), and in cases of the atrophic type may be only present in traces or be absent. Mucin and organic acids such as lactic and butyric acid are generally found.

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The ferment activity, as shown by the rennin and pepsin estimations, is usually much diminished, and in atrophic cases may be entirely wanting. The secretory functions of the stomach may be estimated by the fractional test-meal, in which a pint of prepared gruel is taken, and portions of the gastric contents are withdrawn every quarter of an hour, and a continuous record made of the analytical results.

An **X-ray examination** of the stomach after the taking of an opaque meal will reveal the existence of associated dilatation, atony, or excessive peristalsis, and will be of value in excluding gross organic lesions such as ulcer or new growth.

A **blood-examination** will indicate any associated anæmia such as chlorosis or early pernicious anæmia.

Gastrosocopy is a form of examination associated with some risk to the patient from injury to the œsophagus or stomach, and is not advised as a method of investigation.

It is important that a careful examination of every system be made, so that all possible causes of secondary chronic gastritis may be excluded.

Prognosis.—Primary chronic gastritis is amenable to treatment when its causation has been established and the remedies are directed on the right lines. Old-standing cases often require a long course of treatment before much improvement results, and the practitioner, while giving a hopeful prognosis as regards the ultimate recovery, should exercise caution in forecasting the probable progress of the case. He must also bear carefully in mind the possibility of some grave organic lesion such as early gastric carcinoma. The existence of such a lesion should be suspected when appropriate treatment is not followed by continued improvement.

Treatment.—Careful inquiry should be made into the history of each case from the point of view of diet and mode of life. Often the cause can be found in hurried meals and imperfect mastication, or perhaps, in cases of nervous type, in worry and overwork. A carefully arranged dietary, with regular times for meals and avoidance of overwork, will often rectify the disturbed digestion and be all the treatment required. Physical exercises and recreations, such as walking, riding, golf, etc., are of value.

In many cases a systematic course of treatment will be necessary.

Diet.—The meals should be light, and taken at regular times. Excess of starchy foods and indigestible articles, such as pastry, and foods containing much fat, such as grilled foods, are to be avoided.

Alcohol and tobacco must be prohibited.

When there is gastric fermentation associated with flatulence, it is well for the meals to be taken dry with a minimum of liquid, a glass of hot water or milk being taken two hours after a meal, e.g. at 11 A.M. and 5 P.M., and also in the early morning and at bedtime.

In cases associated with general weakness, loss of weight, and malaise, complete rest in bed is essential, and the diet should consist of milk or citrated milk (2 gr. of citrate of soda to the ounce) given at two-hour intervals, 6–8 oz. at a time. As the gastric symptoms disappear a modified Weir-Mitchell treatment may be instituted.

When solid food can be taken the dietary may be made up of boiled or poached eggs, boiled or steamed fish (sole, plaice, cod, turbot, halibut, fresh herring), chicken, tender mutton, cream cheese, clear soup or beef tea, milk, weak tea, toast, rusks, stale bread, mashed potatoes, purée of spinach, custard or thin milk puddings, and jellies. Fruit is best taken cooked, though fresh orange or grape juice may be allowed.

Gastric lavage is one of the most efficient remedies, and should be employed in cases where the symptoms do not clear up quickly under treatment. The stomach is washed out by means of a soft stomach-tube with tubing and funnel attached, warm water (at about 80° F.) being used, to which bicarbonate of soda, $\frac{1}{2}$ dr. to the pint, may be added if much mucus is present. The irrigation is continued until the washings come away clear, and the stomach is emptied before withdrawal of the tube. In cases with much fermentation, antiseptics such as compound glycerin of thymol solution or listerine, $\frac{1}{2}$ oz. to the pint, may be added to the irrigating fluid. Lavage may be performed once daily for a few days, and is usually followed by great relief from the symptoms.

Massage is of benefit when there is considerable atony of the stomach with dilatation. An X-ray examination will give the best indication for its use.

Spa treatment.—When the gastritis is associated with constipation and intestinal disorder, and also when overwork is an important factor, complete change, and suitable spa treat-

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ment such as may be obtained at Harrogate, Buxton, or Llandrindod Wells, are found very beneficial.

Medicinal treatment.—Reliance should be placed rather on the foregoing measures than on medicines. These are of use—

(1) *To correct defective secretion.*—Dilute hydrochloric acid, 20 drops, taken after meals with some suitable flavouring carminative, is of value; to this may be added the glycerin or wine of pepsin, or a preparation of papain, but it is doubtful if these ferments are of any service.

(2) *To allay fermentation and irritation.*—Carbonate of bismuth and bicarbonate of soda, 20 gr. of each, with 10 min. of glycerin of carbolic acid, to 1 oz. of peppermint water, may be given three times daily; 3 min. of dilute hydrocyanic acid may be added when there is much irritation. Capsules containing phenol 1 gr. and oil of peppermint 1 min. are often of value, or thymol or menthol in 1-gr. doses may be given in pill form thrice daily.

Constipation must be avoided and treated by saline aperients or other suitable remedies.

When recovery is established, prophylactic measures, such as care in diet, exercise, and the general attention to hygiene, are of great importance as preventing a recurrence of the trouble.

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GASTRODYNIA (see STOMACH, FUNCTIONAL DISORDERS OF).

GASTRO-EOTASIS (see STOMACH, FUNCTIONAL DISORDERS OF).

GASTRO-ENTERITIS IN INFANTS (see DIARRHEAL DISORDERS OF INFANTS).

GASTROPTOSIS (see VISCEROPTOSIS).

GASTRORRHAGIA (see HÆMATEMESIS).

GASTROSCOPY (see ŒSOPHAGOSCOPY AND GASTROSCOPY).

GASTROSTAXIS (see HÆMATEMESIS).

GASTRO-TETANY (see TETANY).

GAUCHER'S DISEASE (see ANÆMIA).

GENERAL PARALYSIS OF THE INSANE (Dementia Paralytica).—An affection of the nervous system associated usually, and perhaps always, with infection by the *Spirochæta pallidum* (*Spirochæta pallida*).

Etiology.—Syphilis is the most important of the causative agents of general paralysis.

A clinical history of syphilis is found in a very large number of cases; Wassermann's reaction is positive in a great majority, and in some few the treponema has been found in the central nervous system after death. Some authorities regard syphilis as a necessary factor in the production of general paralysis, and allege that without it there can be no manifestation of this disease; nevertheless, there have been cases in which symptoms, course, and anatomical appearances were indistinguishable from those of general paralysis, but in which there was no history of syphilis and Wassermann's reaction was negative. It is a rare sequel of syphilis, and it is open to doubt whether the adequate treatment of syphilis wholly or in part prevents the appearance of general paralysis. What the adjuvant or accessory causes which assist the development of the disease really are is not at present known. Some allege the deleterious effect of a surplussage of antibodies produced with or without the stimulation of treatment; others, the action of a variety of the spirochæta particularly inimical to nerve tissues; others, a special vulnerability on the part of the nerve tissue; while others, again, enlist the assistance of factors now to be mentioned, and which, before the connexion of syphilis with general paralysis was fully realized, were regarded as of the first importance.

Statistically, it would appear that the incidence of general paralysis is proportional to the degree of civilization reached by a community. Its incidence is particularly upon the educated, and upon those living in a city rather than upon those in the country. Males are affected far more frequently than females, especially among the educated. It appears, as a rule, between the ages of 30 and 50, and some ten to fifteen years after the contraction of syphilis. It occurs but seldom in youth or in old age. Alcoholism, sexual excess, and mental and physical over-exertion have all been adduced as potent contributory agents. It is quite possible that they may be, but it should be recollected that all may occur, separately or together, as aberrations from the normal conduct of the particular individual—that is, as initial symptoms. Traumatism, especially head injury, has been put forward as a cause, and it is credible that injury or shock may make patent hitherto latent symptoms, or may accentuate existing symptoms. Hereditary influence may be dismissed as unimportant.

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Symptomatology.—It is customary, in describing general paralysis, to divide its course into stages. These are so artificial that it would appear better, after describing initial manifestations, to take each possible symptom and indicate its progress. Some of these symptoms never make their appearance, or are early manifestations in some cases and late manifestations in others. The onset of symptoms is usually very insidious, and, even in those cases where the disease has apparently started with a fit or with some acute symptoms of mental disorder, it is generally possible to gather, from a history of the patient's recent life, deviations from the normal. He may have complained of a diminution of his mental energy, or have vaunted in some unusual way its exuberance. His memory may have somewhat failed. Some change in his disposition may have been noted: he may have become irritable or impetuous, or unusually depressed or anxious, or vigorous or buoyant. If he is examined at this stage it may be noted that there is some alteration of expression, and exaggeration or diminution of tendon reflexes. He may complain of headache, neuralgia, gastric disturbances, and insomnia.

Quite commonly, general paralysis is ushered in by symptoms clinically indistinguishable from those of neurasthenia (q.v.). It is always a matter for serious consideration whether such symptoms occurring in one who has had syphilis are likely to be of a passing character or the early manifestations of general paralysis. Even abnormal physical signs may not suffice to distinguish neurasthenia from the early neurasthenic stage of general paralysis, since such signs as sluggishness of the pupils and diminution of tendon reflexes are occasionally met with in cases in which the subsequent history has precluded serious organic disease. A positive Wassermann reaction and a lymphocytosis of the cerebro-spinal fluid suggest general paralysis, but do not infallibly differentiate between it and a syphilitic affection of the nervous system that does not eventually develop into general paralysis.

From this stage onwards, some or all of the symptoms and signs now to be described make their appearance. The pupils are usually unequal and irregular, sometimes small and sometimes very large, but more often of medium size, and they generally fail to react to light. It is to be remarked that this Argyll-Robertson pupil has been noted in alcoholism and other intoxications, in nerve exhaustion,

and in dementia præcox. Ptoxis, strabismus, and optic atrophy, with the concomitant symptoms of diplopia, giddiness, and diminution of vision, may occur.

Speech may be very early affected. Its alteration is in part due to faulty co-ordination and in part to tremor. Words and syllables, and even the elementary sounds composing them, are reduplicated, or unduly separated in time, or hurried and run together. The voice is pitched rather higher than is normal to the individual, and is monotonous, scanning, and drawling. As time goes on, speech becomes more and more unintelligible, until at last the patient emits babbling sounds resembling a baby's first efforts at speech.

Muscular power is not much, if at all, impaired in early stages, and, indeed, the psychic activity of the patient often suggests that he is stronger than he was formerly. Nevertheless, there is much evidence, even in early stages, that the musculature is affected. Tremor is a common symptom. When the tongue is protruded it is at first motionless, but soon two sorts of movement develop—one which, though of small excursion, displaces the whole tongue in an irregular, aimless fashion, and another which consists in fibrillary, rippling movements visible beneath the mucous membrane. These movements become more and more obvious as effort to keep up a position is prolonged. Similar movements may be observed in the muscles of the face and of the limbs. The gait in early cases is often uncertain and stumbling; as time goes on it may become typically ataxic or spastic. After apoplecticiform and epilepticiform attacks, transient palsies of monoplegic or hemiplegic distribution are common, and, even apart from fits, palsies occasionally occur. Spasmodic movements of limbs, grinding of the teeth, and chewing and swallowing movements are frequent. With the progress of the disease the patient wastes and grows more and more feeble, till he becomes too weak to rise from his bed.

Writing is noticeably affected. In the early stages there may be only an unwonted degree of faulty spelling; later, letters, words, and phrases are repeated or omitted. The letters, which in the first few lines may be well formed, gradually get more shaky and sprawled out, so that eventually they cannot be read. Sensation is in many cases blunted in all its forms, but its investigation in the insane meets with difficulties which are often insuperable. Head-

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aches, neuralgia, and paræsthesiæ are frequent. The tendon reflexes are sometimes exaggerated and sometimes diminished or absent. They are generally exaggerated in limbs which have been temporarily or permanently palsied as the result of cerebral attacks. In such cases also the plantar response will probably be extensor. On the other hand, in cases associated with tabes the tendon reflexes are absent. The organic reflexes are usually not affected until late in the progress of the disease, when the patient becomes wholly incontinent of urine and fæces, but much earlier he may occasionally be wet and dirty owing to his loss of sense of the proprieties. Trophic disturbances show themselves in affections of the skin and its appendages, such as vitiligo, bullous eruptions, degeneration and exfoliation of the hair, in the condition of the nails and teeth, in perforating ulcers and bedsores, and in arthropathies and the spontaneous fracture of bones.

Vaso-motor disturbance may be observed in cerebral "congestive attacks," very frequently accompanied and succeeded by temporary paralysis, and in attacks of chills, pallor, and local asphyxia. Some ascribe the extravasations of blood in the lobes of the ears, known as hæmatoma auris, to vaso-motor disturbance. Evidences of arterial and myocardial degeneration are frequent. The changes in the blood are those of chlorotic anæmia.

The *Wassermann reaction* is generally positive in the blood-serum, but it is examination of the cerebro-spinal fluid that gives us the most important information. Here the reaction is probably invariably positive, except perhaps during long remissions, and can very rarely be made negative even by intense anti-syphilitic treatment. The fluid also contains an excess of lymphocytes and an increased amount of albumin. Its condition in general paralysis is consequently similar to that found in tabes.

The tongue is often dirty. The appetite is frequently voracious, and the patient may choke in his hurried efforts to stuff himself. The bowels have constantly to be attended to, since the patient himself usually neglects them. Muco-membranous colitis is not uncommon. The urine at times shows a trace of albumin, and the amount of urea excreted is sometimes diminished. These facts may be correlated with the condition of red granular contracted kidney not infrequently found at autopsy.

The patient's state of general nutrition

gradually deteriorates. At about the middle stage of the disease he tends to get fat, but thereafter flesh is gradually lost until, towards the end, emaciation is extreme. Fits are an important feature. In some cases a seizure is the first morbid phenomenon noticed, and the slight preliminary symptoms recorded above are only recalled after its occurrence; in other cases the fit is the fatal blow which ends the course of the disease, but in the majority fits occur during a middle period. At times they resemble epileptic and at times apoplectic seizures, though the paralysis and aphasia left after the latter type are in most cases of very short duration. The temperature sometimes goes up at the time of the fits; at other times it tends to be below normal.

The **mental symptoms** of general paralysis may be broadly divided into two groups—one including the symptoms of progressive mental impoverishment; the other, accessory symptoms of various kinds which appear here and there during the course of this enfeeblement. Frequently the earliest symptom of mental decadence is a loss of refinement. The finer flowers of good manners are the first to wither, and the patient becomes neglectful in his conduct towards others and of his own clothes and person. His dress may show signs of negligence, and lack of attention to the hair, the beard, the nails, or to his linen may attract attention. Graver defects of memory and intelligence then disclose themselves, and it may be noticed that the most skilled acts of his handicraft or profession are less accurately performed. The patient may indulge in childish acts, and take a childish pleasure in them. The powers of the will, of memory, of judgment, and the affections all diminish. The patient may incompletely realize his circumstances, or, indeed, his own personality. He becomes indifferent to relatives and friends, and loses all interest except in his immediate comfort and his food. His power to perform even the simplest acts diminishes, and eventually, with scarcely a sign of mental life, he becomes bedridden and incapable even of feeding himself.

Some patients present no other mental symptoms than those of progressive mental deterioration, but the majority display symptoms accessory to it. Among these are moral defects quite foreign to the character of the individual when well. Sexual self-restraint may be so far lost that acts of immorality and sexual crime may be committed shamelessly.

GENERAL PARALYSIS OF THE INSANE

The patient may lie, or thief, or commit assaults upon slight or upon no provocation. In business he may buy or sell or speculate in transactions of doubtful honesty which, in their silliness and ineptitude, betray mental enfeeblement. Early in the disease the various mental functions appear sometimes to be stimulated, and memory, imagination, and capacity for work seem greater. Likings and dislikings are exaggerated, and tend to become absurd in the shape of love for unworthy persons or hate of persons quite harmless and even well disposed. The sense of well-being may be exaggerated exceedingly, and the patient become ridiculously proud of his personal appearance, his mental, muscular, and sexual powers, his social station and his possessions. He may generously distribute riches, places, and titles, or, on the other hand, he may be aggressive, quarrelsome, and violent. The folly of his pretensions, which may occupy any grade up to that of godhead, is not apparent to the patient, and he sees no incongruity between them and his immediate surroundings.

Though this expansive grandiosity is very common in general paralysis, there are many patients who present very different symptoms. It has already been mentioned that one of the preludes to general paralysis may take a neurasthenic appearance; this state may become more distinctly one of depression, and the patient may develop those delusions of unworthiness, of guilt and damnation, of financial and social ruin, or of transformation of body which are so common in the syndrome known as melancholia. In some cases exaltation and depression are curiously intermingled: for instance, a patient may deplore that his brain has been turned into millions of bank-notes; or the two states may be clearly divided but may alternate with one another. The symptoms of general paralysis may also simulate systematized delusional insanity or paranoia, but may be differentiated therefrom by the presence of abnormal physical signs and early evidence of mental deterioration. It occasionally presents the clinical picture of confusional insanity; but physical signs and an examination of the cerebro-spinal fluid should suffice to distinguish the one from the other. Hallucinations but rarely occur. Impulsiveness is more common, and may result in assault or acts of sexual violence.

The course of general paralysis is very

variable. It may be rapid in onset and progress and terminate in a few months, or the regular march of symptoms may be very slow; or long periods of remission, when progress is extraordinarily slow or checked, may occur, so that many years may elapse between the first appearance of symptoms and the fatal termination. Usually a case lasts between two and four years.

General paralysis occasionally occurs during juvenility. It can then be attributed to inherited syphilis. Females are as frequently attacked by this form of general paralysis as males. The somatic symptoms and signs are those of the adult variety, but the mental symptoms as a rule consist only of progressive deterioration, without the excitement, or depression, or delusions or fits which are characteristic of the adult variety.

Morbid anatomy.—The dura mater is thickened. Immediately below it there may be layers of bloodclot; that next to the dura and of longest standing may be thoroughly organized, and that next to the pia-arachnoid may consist of quite recently extravasated blood, while the layers intermediate may show more or less organization. The pia-arachnoid also is thickened, in places opaque, and in places hyperæmic. Below it and between the gyri is an excess of turbid and in some cases puriform exudate. Adhesions occur between the pia and the surface of the brain, so that on removing the former the latter is torn. The meningeal and cortical vessels are thickened. The thickening of the meninges consists in part of proliferated fibrous tissue and in part of round-cell infiltration. The brain itself is diminished in weight. Its convolutions are wasted, and the sulci between them are broader and deeper than normal. This condition is most pronounced in the frontal region. The surface of the brain and the ependyma are often sprinkled with white granulations consisting of connective tissue and neuroglia. Microscopically, the cortical cells are irregular in shape, pigmented, and degenerated, with chromatolytic changes, eccentricity of the nuclei, and disappearance of Nissl's granules. The total number of cells is diminished. The tangential fibres immediately below the pia are diminished in number or degenerated. While the cells of higher function are quantitatively diminished, there is hypertrophy of the neuroglia cells and their processes. Spider-like cells with sprawling processes make their appearance, and their fibrillæ

form a dense network. These changes are most noticeable in the neighbourhood of the small vessels. The sheaths of these contain collections of small round cells—"plasma cells"—which are held by some to be pathognomonic of general paralysis. These variations from the normal are widely scattered. They are mostly found in the frontal region, but analogous changes are met with in the cells, cell-processes, neuroglia, and vessels of the corona radiata, optic thalamus, and nuclei of the base, medulla, and spinal cord.

Bacteriology.—Many and various micro-organisms have been described in connexion with general paralysis. None has met with any wide acceptance as the causative factor of the disease. Within the last few years the *Spironema pallidum* has been found in the meninges and brain, and it may be safely anticipated that it, and it alone, will emerge as the veritable exciting agent of this disease.

Treatment.—Prophylaxis lies in the avoidance of the infection of syphilis. If syphilis has been contracted, its thorough treatment and the practice of a hygienic life are indicated. It is widely, though not universally, held that antisyphilitic remedies are not only useless but are harmful when general paralysis is established. Salvarsan, in this condition, is still on its trial, and there is at present but scanty evidence that its use, by either the intravenous, intramuscular, intrathecal, or intracerebral method, has even temporarily relieved symptoms. It is certain that in some cases its administration has had disastrous results. Nucleinate of soda is another drug which has been vaunted. It should be remembered, in estimating the value of this or that mode of treatment, that abatement and remission of symptoms occur quite commonly among those who have had no special treatment, and that before the success of treatment can be established long periods of time must be allowed to elapse. Though there can be but little hope of saving life or mind in any case in which a diagnosis of general paralysis has been arrived at, there is but little doubt that by careful attention to general health, and by the regulation of rest, of exercise, and of sleep, physical and mental decadence can be retarded. Whether a patient should be certified must depend upon whether his mental symptoms are such that he and those about him must be guarded from the effects of foolish, violent, or criminal behaviour, and whether adequate medical and nursing

attendance can be provided elsewhere than in an asylum. Close supervision by physician and nurse is imperative; and in the last stage, when the patient is bedridden, the functions of his bowels and bladder must be regularly watched, bedsores must be prevented, and food must be given carefully in case some should enter the respiratory passages. At regular intervals the patient should be turned from side to side to prevent pulmonary congestion.

E. D. MACNAMARA.

GENU VALGUM (*syn.* Knock-knee).—A deformity in which the line of the tibia is oblique instead of vertical, so that when the knees are together and the patellæ directed forwards the internal malleoli are separated.

Etiology.—The common varieties of genu valgum are (1) due to rickets, occurring in young children, and (2) adolescent, commencing between the ages of 12 and 18 years. In *rickets* there is a general laxity of ligaments, and the internal lateral ligament of the knee becomes stretched; an abnormal amount of weight is then transmitted through the outer part of the knee; the inner condyle of the femur, and the internal tuberosity of the tibia, being relieved of weight, grow to an abnormal extent. In the *adolescent* form, the muscles are weak and toneless, owing to increase in the body-weight, and to anæmia and toxæmia from some focus of latent sepsis, such as chronic tonsillitis and adenoids, or constipation. The patient rests the muscles by making use of the ligaments, developing a habit of standing with knees extended and feet apart; the internal lateral ligament is stretched, and the external condyle, transmitting an undue amount of weight, atrophies.

Less common causes are:

i. *Trauma*—division of the internal lateral ligament, fracture or separation of the lower epiphysis of the femur, fracture of the tibia, dislocation of the knee, and riding. Sometimes the deformity is produced by faulty splinting, especially by the prolonged use of a double Thomas hip-splint without proper precaution, and during the treatment of a fracture by slinging the splinted leg in such a position that there is strain on the internal lateral ligament.

ii. *Chronic inflammation*—osteo-periostitis or cancellous osteitis of the tibia, osteo-arthritis of the knee.

iii. *Paralysis of the thigh muscles.*

Pathology.—There are two common vari-

GENU VALGUM

eties of bony deformity: (1) Overgrowth of the internal condyle, with atrophy of the external condyle of the femur and the outer tuberosity of the tibia owing to the outward displacement of the line through which the weight is transmitted; the drag on the internal lateral ligament causes periostitis in the neighbourhood of its insertion in the inner tuberosity, and this becomes hypertrophied from hyperæmia; the patella is displaced laterally. (2) Angulation of the shaft of the tibia or femur, as a result of injury or inflammation.

Flat-foot and genu recurvatum are often associated with genu valgum, and in many of the rickety cases the deformity is compensated by a bowing of the lower end of the tibia, owing to the child walking with inverted feet; this compensatory bow-leg should on no account be corrected, or the genu valgum would be increased.

Symptomatology.—There is aching pain, especially with fatigue, and the patient is apt to fall in running, owing to the knees striking one another. A small child is often brought for treatment because it is noticed to walk with the feet turned in ("pigeon-toe gait"), thus bringing them under the centre of gravity of the body; in adolescents, on the other hand, flat-foot of the second degree is usually present. On examination it is seen that, with the knees together and the patellæ directed forwards, the internal malleoli are separated; if the deformity is due entirely to changes in the femur, it disappears when the knees are flexed, but if there is pronounced angulation of the tibia, the deformity still is obvious. When the child lies down, it is seen that the deformity is lessened as the strain is removed from the internal lateral ligaments; in this position the laxity of these ligaments can be demonstrated by passively abducting the legs, with the thighs fixed and the knees extended.

Prognosis.—In all cases a good result is to be obtained from careful conservative or operative treatment.

Treatment.—General treatment includes the use of cod-liver oil or other tonic, and the eradication of all foci of infection.

In small children suffering from rickets it is sufficient to treat the general disease, and to keep the patient off the feet by means of long wooden splints, extending from the upper part of the thighs to well beyond the feet; thighs and legs are massaged daily, and are manipulated by pressing the lower end of the

GENU VARUM

thigh outwards with one hand and the lower end of the leg inwards with the other.

At a later age, it is enough in slight cases to order physical exercises, and to correct faulty habits of standing and walking by training the child to walk with feet turned in or parallel, the inner side of the soles and heels of the boots being raised; these children wear out their boots very rapidly, so it is useful to have a thin rubber sole and heel added.

In more advanced cases irons should be ordered, in the form of a Thomas knee-brace or some modification. An external bar extends from the great trochanter to the heel of the boot, and a posterior bar reaches from well above to well below the knee; the deformity is corrected, and the joint is extended, by means of a genu-valgum cap, and the foot is inverted by a valgus T-strap and by raising the inner side of the sole and heel of the boot.

The test of recovery is to examine the extended knee for lateral mobility; splints must be employed as long as passive abduction can be produced owing to laxity of the internal lateral ligament.

Any form of iron which has a joint at the knee is useless, and padded external wooden splints extending from thigh to leg, and allowing walking, do not exert sufficient leverage to be of any value.

In children over five, if there is more than 3 in. of separation of the malleoli, and no active osteitis or rickets is shown by X-ray examination, and in adolescents with more than 4 in. of separation, the deformity may be corrected by operation. The simplest operation, only suitable for young children, consists in forcibly correcting the deformity by wrenching with the hands or by means of an osteoclast, a greenstick fracture of femur or tibia or separation of epiphysis being produced. For older children or adults, open osteotomy is performed. After operation, the deformity should be slightly over-corrected, and the limb fixed in plaster of paris for six weeks as soon as the wound is healed, care being taken to avoid hyper-extension of the knee. For a year afterwards; knock-knee irons or Thomas calliper splint should be worn, the limbs being massaged and exercised.

C. W. GORDON BRYAN.

GENU VARUM.—A deformity in which, in bilateral cases, when the patient stands with the internal malleoli in contact, the knees are separated. It is usually associated with curvature of the tibiæ, and sometimes with

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curvature of the femora, these two factors accounting for the greater part of the bowing of the limbs.

Etiology.—At birth there is often some bowing of the legs, as a result probably of the position in utero; during the first year the legs gradually grow straight, without treatment.

The commonest cause of genu varum is rickets; the deformity sometimes develops before the child walks, owing to a habit of sitting in the "tailor's position," with the legs crossed, the soft tibiae becoming bent at their centre with the convexity outwards; when the deformity commences after walking, the outward bowing of the tibiae is usually associated with a more acute angulation, with the convexity forwards.

Sometimes there is genu varum in one limb and genu valgum in the other; in the case of an infant with rickets it has been supposed that this combination is due to the child being habitually carried on the same arm, but when the deformities develop in later childhood they are difficult to explain.

In older people, especially during adolescence, riding produces genu varum, and malnutrition is here a contributory factor.

It is common for genu varum to develop after middle age, as a result of osteo-arthritis or in association with osteitis deformans.

Traumatic genu varum may be produced by injuries in the region of the knee, particularly fracture or separation of the epiphysis of the lower end of the femur, fracture of the upper third of the tibia, and dislocation of the knee-joint. It sometimes follows suppurative arthritis of the knee.

Pathology.—The outer part of the capsule and the external lateral ligament of the knee are lengthened, and there is deformity of the tibia and femur; the outer condyle is increased from above downwards in cases due to rickets. As time goes on, the crucial ligaments become stretched, and the knee is unstable; it is subject to attacks of synovitis, and osteo-arthritis is a common sequela. Genu recurvatum is often present in cases of traumatic origin.

Symptomatology.—Children are usually brought for treatment because of the obvious deformity and peculiar gait; they walk in a waddling or rolling fashion, which closely resembles that of congenital dislocation. In severe cases there is shortening of the calf-muscles, and the child walks on the toes, which are turned inwards. Pes valgus and pes planus are frequent complications. In later life the

laxity of ligaments causes the knee to be unstable and to give way on sudden movements, and symptoms result from recurrent attacks of subacute synovitis, or from osteo-arthritis.

Diagnosis.—It is important not to mistake a case of genu valgum, with compensatory curve of the lower third of the tibia, for genu varum; correction of the tibial curve in the former condition would lead to aggravation of the genu valgum. In a case of bow-legs, it must be decided whether the deformity is limited to the tibiae, or if curvature of the femora coexists; if the former, when the patient stands with the malleoli in contact the knees can be brought together.

Prognosis.—In infants and in children under four complete recovery may be expected from careful treatment, but in cases of considerable deformity in older children it is difficult to obtain limbs of good alignment without upsetting the mechanism of joints and ligaments and weakening the limbs; in no case should operation be undertaken without careful consideration of all the factors.

Treatment.—In cases seen during active rickets the child must be kept from standing, or from sitting with the legs crossed, by means of long splints applied to the inner aspect of the thighs and legs and projecting well beyond the feet; the knees are firmly bandaged to the splints so as to correct the deformity. The splints are removed twice a day to allow of the limbs being massaged and forcibly manipulated; the knee is pressed inwards, the lower part of the leg outwards.

In older children with a slight degree of genu varum an internal iron may be used, extending from the upper part of the thigh to the boot, with a knee-cap pulling the knee inwards; there should be no joint at the knee. In the absence of pes valgus and pes planus, a varus T-strap may be used, the outer part of the sole being thickened, but care must be taken not to produce pes planus by these methods.

In severe cases, usually in children over four, the deformity may be corrected by operation, the tibia being fractured at the summit of the curve by forcible manipulation, by means of an osteoclast, or by open osteotomy, according to the circumstances of the individual case. It may be necessary also to divide the tendo Achillis and to perform an osteotomy of the femur. After operation, the limbs must be fixed in plaster of paris for at least a month.

C. W. GORDON BRYAN.

GLANDERS

GEOGRAPHICAL TONGUE (*see* STOMATITIS AND GLOSSITIS).

GERMAN MEASLES (*see* RUBELLA).

GIDDINESS (*see* VERTIGO).

GIGANTISM (*see* PITUITARY GLAND, AFFECTIONS OF).

GILLES DE LA TOURETTE'S DISEASE (*see* TICS AND HABIT SPASMS).

GINGIVITIS (*see* STOMATITIS AND GLOSSITIS; ORAL SEPSIS).

GLANDERS (*syn.* Farcy).—A somewhat rare acute or chronic infective disease of a granulomatous type, malignant in character, with grave constitutional symptoms, specific inflammatory lesions of the nasal and respiratory passages, lymphatic vessels and glands, and characteristic cutaneous eruptions.

Etiology.—Glanders is due to the *Bacillus mallei*, and is communicated to man from members of the equine family—horse, ass, and mule. Its incidence is chiefly among coachmen, grooms, stable-boys, equestrians, and veterinary surgeons, persons whose activities bring them into close contact with these animals. It may be communicated from man to man, and has been contracted from cultures of the specific organism in the laboratory. The virus usually gains admission through an abrasion of the skin or mucous membrane of the mouth, eye, nose, or other part of the respiratory tract; but it may enter through these structures without any visible breach in their continuity, as, for instance, by the hair-follicles. This is more likely to happen if the virus is mixed with oleaginous matter (as in the discharge from a glanderous abscess of a lymph-vessel—a “farcy bud”) and aided by friction. It may be communicated by inhalation of the spray produced by the sneezing of an infected animal.

Pathology.—The most prominent feature of glanders in man is the formation of hard, shotty tubercles produced by an infiltration of leucocytes and young connective-tissue cells. Typical giant-cells are not present. In the skin these nodules resemble somewhat those of the papular stage of variola. They vary in size from a millet seed to half an inch or more in diameter. Before becoming elevated above the skin surface they are of a dark-red colour, and when large may resemble erythema nodosum. Afterwards they swell up, assume a yellowish colour owing to coagulation necrosis, and resemble pustules having

an inflammatory areola. They do not become umbilicated, and contain not pus but a solid granulomatous mass. They lie in cup-shaped cavities in the corium, and, although their contents have become necrosed, the epithelium over them appears healthy, as if it were better able to withstand the toxins of glanders than the cutis vera itself. Later on a vesicle appears on the summit of each nodule. Eventually, if the patient has not succumbed, the vesicles rupture and the contents are discharged as a puriform-looking substance consisting of “not pure pus, but a lymph containing a mixture of blood from the peripheral vessels, glanders bacilli, a few pus-cells, and many rags of tissue” (Unna). If the general infection does not rapidly cause death, the resulting ulcers, partly by coalescence and partly by spreading peripherally owing to the growth of bacilli in the lymph-vessels, become the extensive serpiginous ulcers with undermined edges which characterize chronic glanders.

Symptomatology.—The symptoms vary according to the site and intensity of the local lesion, and the distribution and number of the metastatic foci. Generally speaking, the virulence of the infection is inversely proportional to the length of the period of incubation, which varies from three days to six weeks. During incubation there may be no symptoms, or at most a feeling of depression.

Cases may conveniently be divided into (a) those with *local*, (b) those with *general infections*, and the general infections again may be divided into *acute* and *subacute* or *chronic* cases.

If the infection is very mild the disease may remain *local*, but usually general infection follows sooner or later, and even when healing does occur there is a tendency to break down again. With purely local disease there may be little constitutional disturbance.

When *general infection* takes place in *acute* cases, the period of invasion is ushered in by malaise, headache, general lassitude, pyrexia, and anorexia, followed by severe pains in the bones and joints, and thus bears a strong resemblance to that of acute rheumatism. *Pari passu* the seat of inoculation becomes inflamed. At the same time the axillæ and groins become painful, and slight enlargement of the lymphatic glands of these regions may be noticed. The pulse is full and rapid, the tongue coated and foul, the urine scanty and high-coloured. A little later a characteristic varioloid eruption appears. At first small red spots resembling flea-bites show themselves, becoming

GLANDERS

first papular and afterwards vesicular. The vesicles acquire a pseudo-pustular appearance, the tubercles being of a yellowish colour in the centre and surrounded by a red areola. Ultimately the epidermis gives way and ulceration follows, leaving small holes filled with necrotic material. In some cases the eruption takes a bullous or pemphigoid form, the flattened bullæ containing a sero-sanguineous liquid; these ultimately break down, frequently coalescing to form large irregularly shaped ulcers covered with soft brownish-coloured sloughing surfaces. When about the head and neck they are surrounded with an erysipelatous redness, and considerable œdema of the skin and subcutaneous tissues. When occurring more deeply, glanderous tubercles may form diffused swellings of considerable size, which by undermining the skin may cause much sloughing of the integument and deeper structures. In many cases, as the disease advances, the mucous membranes of the mouth and respiratory passages become affected. Tubercles appear in the nose, frontal sinuses, trachea, and bronchi, as well as in the lungs and pleura, there giving rise to pneumonia and pleurisy. The nasal discharge at first is thin and clear, but later thick, sticky, and sanguineo-purulent. Toxic albuminuria, delirium, a high oscillating temperature, rigors, and diarrhoea frequently supervene. The patient then soon sinks into an adynamic or typhoid state, becomes comatose and dies of exhaustion, usually well within three weeks from the date of the inoculation.

In the *subacute or chronic* form the incubation period is far longer than in the acute form, and the general systemic disturbance is much less marked. The tubercles and resulting abscesses are slower in development, and when ulcers form they are frequently of the ecthymatous type, very persistent and intractable, and often penetrating to the bone. The nasal passages, larynx, trachea, and lungs may become involved, and aphonia and hæmoptysis ensue. Glanderous abscesses may form in the kidneys, testes, liver, or spleen, and osteomyelitis and joint-abscesses are not uncommon. The chronic form may become acute, but, more often, the patient succumbs to general cachexia and renal disease. The average duration of chronic glanders is said to be about four months. If the patient recovers, the ulcers undergo a gradual healing and the general symptoms disappear. After an apparent recovery, recrudescence may take place

even a year later, and death ultimately result.

Diagnosis.—In the early stages diagnosis is by no means easy, and the disease may readily be mistaken for acute rheumatism, enteric fever, or pyæmia. The early eruption may resemble that of variola. The fact that it almost always occurs in men who come into contact with horses or their kind should put the practitioner on his guard. When the eruption is fully developed all doubt should be at an end. The nasal discharge, the erythematous flush in the face and eyes, the characteristic tubercles, deep-seated, knotty masses and local suppurations in the subcutaneous cellular tissues and muscles, will confirm the diagnosis. But, without waiting for these clinical appearances, bacteriological and biological methods should be resorted to as soon as suspicion arises. The *Bacillus mallei* should be looked for in any suspected local lesion or nasal discharge. Fluid from unopened abscesses or farcy buds should be taken for cultural purposes. Mallein, in 10- to 15-min. doses, when injected hypodermically into a patient suffering from glanders, produces a marked rise in temperature in twenty-four hours, and an inflammatory exacerbation of the local lesions. The agglutination test may also be used. If agglutination takes place with a 150-fold dilution, the presence of glanders may be assumed. The method of diagnosis by complement fixation, as in the Wassermann reaction, has already given good results.

Prognosis.—Mild, purely local cases, if diagnosed early and appropriately treated, may heal in a few weeks and general infection be avoided. In cases with acute systemic invasion death almost invariably results in from two to six weeks. The chronic form is fatal, sooner or later, in almost 50 per cent. of the cases.

Treatment.—The focus or foci of purely local glanders should be excised if practicable, and the resulting wound cauterized with a 10-per-cent. solution of phenol, and left open to heal by granulation. In acute glanders, after dealing surgically, if possible, with the primary lesion, the only available treatment is that directed towards alleviating the symptoms as they arise, and at the same time maintaining the constitutional vigour of the patient by rest, diet, alcoholic stimulants, etc. Such drugs as quinine, benzoate of soda, and aceto-salicylic acid are beneficial in some cases.

GLANDULAR FEVER

The nasal and other cavities subject to discharges should be douched with a disinfectant such as permanganate of potash, iodine, or formaldehyde, in aqueous solution. In chronic glanders, abscesses and fistulous tracts should be opened up freely and curetted and undermined, and loose integument cut away, the raw surfaces being thoroughly cleansed with 10-per-cent. phenol or 12-per-cent. chloride of zinc. Several repetitions of this curetting and disinfecting may be required, and no attempt should be made to obtain healing till all offending bacteria are destroyed. Hypodermic injections of mallein ($\frac{1}{10}$ – $\frac{1}{5}$ c.c.) every second or third day, according to the reaction obtained, may in some cases do good. An anti-glanders serum has been used, but no noteworthy results have been recorded.

WILLIAM MITCHELL.

GLANDULAR FEVER.—An acute infectious fever, characterized by swelling of the lymphatic glands, especially of the cervical group. It affects children between the ages of 3 and 12, and only rarely attacks adults. The etiology is not known.

Symptomatology.—The disease is rare in this country, but it is probable that mild epidemics and sporadic cases go unrecognized. The *incubation period* is from five to ten days, usually about seven. The *onset* is sudden, with moderate fever, and pain and stiffness in the neck and in the muscles of the trunk and limbs; in the more severe cases the fever may reach 103° or 104° F. with vomiting, abdominal pain, and constipation. On the second or third day of the fever a swelling of the deep cervical glands, usually on the left side, is detected, reaching its maximum in two or three days, after which it slowly subsides. Simultaneously or successively other groups of lymphatic glands, in the axillæ, the groins, and the epitrochlear regions, become enlarged and tender; and pain and tenderness in the abdomen suggest that the mesenteric and retroperitoneal glands also are involved. The spleen and liver are sometimes moderately enlarged. There is no sore throat and no skin eruption. With the subsidence of the glandular swellings the fever ceases, usually in the second week; but the effects of the attack are seen in a prolonged convalescence, often accompanied by a pronounced anemia and considerable loss of flesh. Complications of any kind are rare, and the prognosis is uniformly good.

GLAUCOMA

Diagnosis.—The absence of sore throat, and of a rash, and the curiously protracted fever coincident with the successive widespread glandular swellings, should suffice to distinguish this from other infective fevers; and the occurrence of such symptoms in several patients, especially in a school or in a family, can hardly be attributed to any other disease. The prolonged enlargement of the glands and the slow convalescence should be remembered, since forgetfulness of these points has allowed the mistaken diagnosis of *tuberculosis*, and encouraged an unnecessary operation.

Treatment.—The treatment is that of all febrile ailments—rest in bed, light diet, and aperients. Local applications of heat or of belladonna may be used to relieve the pain of the swellings, and a few doses of a febrifuge mixture, such as the following, may be given:—

R Tr. acon. ℥viii.

Liq. ammon. acet. ℥ss.

Aq. menth. pip. ad ℥ij.

Two drachms to be taken every three hours.

In convalescence an iron tonic and country- or sea-air are beneficial.

HUGH THURSFIELD.

GLAUCOMA.—The distinguishing characteristic of glaucoma is an increase of the intra-ocular tension. This may be measured by palpating the globe, as near the equator as possible, with a finger of each hand (not two fingers of one hand), as the patient looks downwards. Abnormal tension is conveniently, if somewhat conventionally, expressed under the terms plus or minus 1, 2, or 3, the figure 3 representing in one direction stony hardness, in the other an almost complete lack of resistance. More accurate and more comparable results are obtainable by means of the Schiötz tonometer, which indicates that the average normal tension is about 19 mm. of mercury.

In theory, glaucoma might be produced by (a) an increase in the secretion of the intra-ocular fluids, the drainage remaining the same, or (b) a diminution of drainage, the secretion remaining the same. In actual fact, nearly all cases of the disease are referable to the second class—the group of “retention glaucomas.” This occlusion of drainage may originate in structural peculiarities of the globe which are not the result of manifest antecedent disease; or it may be an accident in the course of some other ocular affection. On this basis

GLAUCOMA

a distinction is drawn between (1) primary and (2) secondary glaucoma.

PRIMARY GLAUCOMA

Etiology.—In normal circumstances the intra-ocular tension is maintained chiefly by the transudation of aqueous humour from the vessel-plexuses of the ciliary processes, and by its drainage at the angle between the iris root and the cornea (corneo-iridic angle). Glaucoma is observed for the most part in eyes which are predisposed to its occurrence by certain factors that interfere with this free circulation. One of the most important of these is smallness of the globe, of which the most reliable outward indication is an unduly small diameter of the cornea (under 11.6 mm.

drainage, while the shallowness of the anterior chamber, resulting from the increasing bulk of the lens, renders the corneo-iridic angle more acute and less spacious (Figs. 28, 29). Hence age predisposes to glaucoma; while heredity, and perhaps race, also play a part.

The eye being so predisposed, if a cause of congestion of the ciliary body (emotion, fatigue, constipation, etc.) be added, the ciliary processes may come into contact with the equator of the lens. The freedom of circulation of the aqueous humour from behind forwards will be impaired, the pressure behind the lens raised above the pressure in front, the lens and iris driven forwards, the corneo-iridic angle occluded, drainage obstructed, and the tension raised. Probably also the swollen



Fig. 28.—Normal corneo-iridic angle.



Fig. 29.—Glaucomatous corneo-iridic angle.

horizontally). Smallness of this diameter implies limitation of the space enclosed within the ring of ciliary processes, and of the space between these processes and the lens equator (circumlental space). Again, the size of the lens shows a certain independence of the size of the eye; a lens of normal or even supernormal diameter may be found in a small eye, and moreover the lens continues to enlarge throughout life, whereas the eye ceases to grow after early adolescence; both these factors contribute to produce, in small eyes especially, a narrowing of the circumlental space. A small eye, too, is often, though not necessarily, hypermetropic, and in this case the constant use of the accommodation produces hypertrophy and increase in bulk of the ciliary muscle as a whole, from which again the circumlental space suffers limitation. In old people processes of sclerosis at the corneo-iridic angle interfere with the freedom of

ciliary processes occlude the corneo-iridic angle by exercising direct pressure from behind on the iris root. Should the congestion now subside, the lymph circulation will be restored, drainage will be re-established, and the tension will fall; if, however, the attack be prolonged, the contact of the iris root with the cornea, at first a mere apposition, may become a definite adhesion, and unless artificially freed will permanently hinder the restoration of drainage.

Symptomatology.—The signs and symptoms of *acute* glaucoma are all deducible from the raised tension. Interference with the circulation causes superficial congestion of all the episcleral vessels in acute, of the veins more especially in subacute, glaucoma. In the more acute forms the lids may be swollen. Impairment of lymph-drainage induces oedema of the cornea, which expresses itself in a general haziness and a roughening and stippling of the surface. The effect upon vision is that of

GLAUCOMA

looking through a piece of glass which has been breathed upon; lights are surrounded by rainbow-coloured haloes, and visual acuity is much reduced. The forward displacement of the lens produces shallowness of the anterior chamber. Compression of the nerves of the sclera and iris results in severe pain, not confined to the eye, but of neuralgic type, and shooting along the distribution of the fifth nerve; its severity not infrequently induces sickness, leading sometimes to a mistaken diagnosis of "bilious attack" with headache, and to a fatal delay in treatment. The innervation of the iris is paralysed, and the pupil is semi-dilated, fixed, and usually somewhat oval.

All these symptoms are capable of immediate alleviation if the increased tension be relieved; if it persist, however, more permanent damage ensues. The sclera begins to give way, and first at its weakest part—the optic-nerve entrance. Glaucomatous cupping of the papilla results, which differs from the physiological variety in extending quite to the periphery of the disc, and from the atrophic in its greater depth and in the abruptness of its edges. The resulting atrophy of the nerve manifests itself by increasing pallor of the disc, and by a constriction of the visual field which begins characteristically on the nasal side, and in its progress may leave only a small area of retained sight round the fixation point; in this case the patient's central vision may be relatively good, but he has much difficulty in getting about; he sees as if he were looking through a long tube of narrow calibre. Finally, vision is totally extinguished, the lens becomes cataractous, and the sclera undergoes stretching in localized areas. When the train of symptoms is as above described, but of less fulminating intensity, the glaucoma is said to be *subacute*.

The recognition of *chronic* glaucoma is more difficult. Congestive phenomena may be totally absent or only occasionally present. Externally the eye may appear quite normal, or there may be a little corneal haze, with perhaps some shallowness of the anterior chamber and dilatation of the pupil. Tension may be only slightly and not constantly raised, and the patient's sole complaint may be of gradually failing vision; not infrequently, however, a history is obtained of temporary attacks of "halo-vision." In this case the diagnosis depends on the discovery of cupping and atrophy of the disc, and on the character-

istic constriction of the nasal field. A form of glaucoma is described in which cupping and atrophy exist although the tension is never appreciably raised (glaucoma simplex).

Juvenile glaucoma (*buphthalmos*) is a variety which is almost always of prenatal origin, and due to congenital malformation of the corneo-iridic angle. It differs from the adult form in that the weaker tunics of the infant's eye undergo a general expansion, especially in their anterior segment. The sclera becomes thin and blue, the cornea enlarged, the anterior chamber very deep, the iris slaty-blue in colouring, atrophic and tremulous owing to insufficient support by the lens, which, of course, does not share in the general enlargement of the globe. There is usually deep cupping of the disc.

Diagnosis.—The most frequent and most fatal error of diagnosis is the confounding of a case of acute glaucoma with one of *iritis*. Common to both are the congestion of the eye without muco-purulent discharge, the neuralgic character of the pain and, not infrequently, the haze of the media, causing the iris pattern to appear dull and blurred. The mistake would usually be avoided if the tension were carefully tested and compared with that of the other eye. Cases of acute iritis with increased tension do occur, but they are rare, and in general it requires no *tactus eruditus* to distinguish the stony hardness of an acute glaucoma from the dimpling under palpation of the normal eye. Of scarcely less importance is the observation of the pupil: in glaucoma it is dilated, often oval, and without adhesions to the lens capsule (*synechiæ*) or fine irregularities; in iritis, provided atropine has not been used, it is small, shows *synechiæ*, and is irregular. The haze of the media in iritis is rather in the aqueous than in the cornea, and the corneal surface is usually smooth. The discovery of exudates in the anterior chamber, or of deposits of cells on the back of the cornea (*keratitis punctata*), would prove the case to be one of irido-cyclitis. Cupping of the disc affords no aid in the diagnosis of acute glaucoma, since it takes some time to develop, and the disc is usually invisible owing to opacity of the media. In *acute conjunctivitis* the presence of purulent or muco-purulent discharge, the clear cornea, normal anterior chamber, active pupil, intact vision and superficial character of the pain should preclude the possibility of error. In the case of chronic glaucoma the danger is that the

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condition may be overlooked altogether, while the patient vainly seeks a pair of glasses to suit his sight; an expert examination of the fundus and field of vision is requisite.

Treatment.—In *acute* glaucoma operative intervention at the earliest moment is imperative. While preparations are being made, hot bathing and leeches to the temple should be used, a purgative should be given, and a 1-per-cent. ointment of eserine (the alkaloid, not one of its salts) should be applied repeatedly to the eye. By these measures the tension can often be reduced and the depth of the anterior chamber increased, so that the operation is undertaken under much more favourable auspices. In order to obtain these conditions it is sometimes justifiable to postpone operation till the following day, a further advantage being that the patient can be prepared properly for a general anæsthetic. The operation of election in acute glaucoma is a broad iridectomy reaching quite to the root of the iris, whereby the adhesion at the corneo-iridic angle is separated and the drainage channels are reopened.

In *chronic* glaucoma the necessity of operation is less urgent. Usually treatment is begun by the use of eserine in the weakest strength which will keep the pupil thoroughly under its influence ($\frac{1}{4}$ –1 gr. of eserine sulphate or salicylate to 7 oz. of distilled water; sometimes even weaker solutions suffice). If eserine causes conjunctivitis or cramp-like pain, as it sometimes does, a solution of pilocarpine nitrate ($\frac{1}{4}$ –1 per cent.) may be substituted. During this course of treatment careful watch must be kept on the tension, field of vision, and visual acuity, and if retrogression is noted operation should be advised. The results of iridectomy are less satisfactory in chronic than in acute glaucoma, and in recent years various measures have been devised which aim at the establishment of a permanently leaking fistula between the anterior chamber and the subconjunctival tissues. Of these the operation of trephining is most in favour; it consists in the excision of a small disc of sclera at the sclero-corneal junction.

SECONDARY GLAUCOMA

In secondary glaucoma the symptoms detailed above may be masked and modified to some extent by the underlying disease.

Etiology.—The following are the conditions to which it most commonly owes its origin:

1. **Iritis and irido-cyclitis.**—Glaucoma may

result in two ways. (a) There is a variety of irido-cyclitis characterized by the secretion of an excess of albuminous fluid, and by clogging of the corneo-iridic angle with leucocytes (serous cyclitis); the tension is raised and the anterior chamber is deep. (b) The formation of posterior synechiæ may so glue the margin of the pupil to the lens as to form a water-tight adhesion; the aqueous humour collecting behind the iris balloons it forwards (*iris bombé*) and causes occlusion of the corneo-iridic angle. To relieve the condition the circulation between the posterior and anterior chambers must be re-established by means of an iridectomy. If possible, the attack of glaucoma should be anticipated by performing the operation before the pupil is completely bound down.

2. **Perforating wounds and ulcers of the cornea.**—The iris becomes entangled in the cicatrix and is dragged forward, while the free circulation of the aqueous humour is impeded. *Iris bombé* may also result from adhesion of the iris to the lens capsule. The iris must be freed from the cicatrix, or the communication between the posterior and anterior chambers restored by an iridectomy.

3. **Dislocation of the lens.** (See LENS, DISLOCATION OF.)

4. **Intra-ocular tumours.**—Much the commonest form is sarcoma of the choroid, which occurs in elderly people. Since the fundus is frequently invisible owing to haze of the media, it may be difficult to distinguish the case from one of primary acute glaucoma. There may be a history of gradual failure of vision before the onset of the glaucomatous symptoms; a combination of glaucoma of short duration with absolute blindness, or with detachment of the retina, is suspicious. Blind painful glaucomatous eyes should usually be excised. Glioma of the retina is a disease of childhood, and the glaucoma which ensues is of the buphthalmic type, but true buphthalmos is seldom very closely imitated.

5. **Intra-ocular hæmorrhage.**—Large effusions into the anterior chamber or vitreous are comparatively seldom followed by glaucoma, especially when they occur in eyes previously healthy, but the sequence is not infrequent in certain forms of profuse retinal hæmorrhage (thrombosis of the central retinal vein). The prognosis in the latter case is bad.

GEORGE COATS.

GLEET (see GONORRŒA).

GOITRE

GLÉNARD'S DISEASE (*see* VISCEROP-TOSIS).

GLOBUS HYSTERIUS (*see* HYSTERIA; PHARYNX, SPASM OF).

GLOSSITIS (*see* STOMATITIS AND GLOSSITIS).

GLOSSO - LABIO - PHARYNGEAL PARALYSIS (*see* BULBAR PALSY).

GLOTTIS, OEDEMA OF (*see* LARYNX, OEDEMA OF).

GLYCOBURIA (*see* DIABETES MELLITUS; PITUITARY GLAND, AFFECTIONS OF; URINE, EXAMINATION OF).

GOITRE.—The term goitre is somewhat loosely used to describe any form of thyroid enlargement. Such enlargements may be divided into three main groups:

1. Inflammatory swellings.
2. Localized tumours.
3. Diffuse enlargements of the gland.

The first group includes rare conditions such as gummata, localized abscesses, and hydatids. Very rarely a diffuse inflammatory enlargement known as acute thyroiditis (Riedel's disease) may be seen; it is more common in the male, is characterized by a rapid enlargement of the thyroid, which is adherent to and compresses the surrounding tissues, and may give rise to urgent dyspnoea.

The last two groups include the majority of cases of enlarged thyroid, and may be divided as follows:—

INNOCENT.

(a) Localized—

Adenomata.
Cysto-adenomata.
Fibro-adenomata.

(b) Diffuse—

Parenchymatous.
Adeno-parenchymatous.
Exophthalmic goitre (Graves's disease).

MALIGNANT.

Carcinoma.

In actual practice the line of distinction between these varieties is not clearly marked. Thus it is found that the simple parenchymatous enlargements are not uncommonly associated with the presence of adenomata. Moreover, all degrees of hyperthyroidism may be seen, from the simple parenchymatous to the true exophthalmic goitre. (This latter variety is considered under EXOPHTHALMIC GOITRE.)

ADENOMATA

These tumours are about ten times more common in women than in men. Except in so far as they are more prone to arise in thyroids the seat of a general enlargement, they show no tendency to be endemic. They are most common between the ages of 20 and 40.

Pathology.—In the early stages there are one or more pure adenomata, appearing as small rounded encapsulated masses which often have a granular solid appearance. In other cases small glistening areas of colloid material may be seen. As the tumour increases the capsule becomes thicker and better defined, the centre at the same time tending to break down from an accumulation of colloid or from hæmorrhage. By this means a cysto-adenoma is formed, and it is rare to find the larger tumours solid throughout. In many cases almost a pure cyst may result. Microscopically, the tumour very closely resembles a normal thyroid. In the solid varieties the vesicles may be largely filled with papillary processes of proliferated epithelium, the so-called foetal adenoma, and in such cases slight hyperthyroidism may be present. In other cases the proliferation is slight, and well-defined vesicles are present. The vesicles may be enlarged and filled with colloid, forming small cysts.

In longstanding cases of multiple adenomata there may be much fibrous tissue, giving rise to the so-called fibro-adenoma.

Symptomatology.—The presence of a tumour which forms a rounded mass in the substance of the thyroid is often the only evidence of a small single adenoma. The tumour is soft and semi-fluctuant, and follows the movements of the trachea during swallowing. If in the lateral lobe, it will be in part beneath the sterno-mastoid muscle. If in the isthmus, it may be confused with a tuberculous abscess, the latter, however, being less globular and more definitely fluctuating. If cystic, the tumour is tense and does not fluctuate, the solid tumours, therefore, often appearing softer and more fluid than the cysts. If multiple, there will be an irregular enlargement of the thyroid. Apart from the presence of the tumour, the most common symptom is that of pressure. The trachea is the first structure to be compressed, the patient suffering from dyspnoea, which is increased on exertion. This symptom is more pronounced if the tumour is growing from the lower border of the thyroid and becomes fixed in the thoracic inlet, the so-called substernal

GOITRE

goitre. Acute dyspnoea may also arise if there is sudden hæmorrhage into the cyst. The veins, or even the recurrent laryngeal nerve, may be compressed, and it has already been mentioned that in certain cases there is a mild amount of hyperthyroidism, seldom sufficient to be designated exophthalmic goitre. It may be noticed that the tumour increases in size during the catamenia or pregnancy.

Prognosis.—If untreated the tumour tends to progress steadily in size, giving rise to more marked pressure symptoms. Rarely it may be the site of malignant changes.

Treatment.—On account of the deformity, the danger of pressure symptoms, and the onset of malignant disease, the tumour should be removed. Whenever possible, the new growth should be dissected out of its capsule. In cases of acute dyspnoea, tracheotomy may be required.

PARENCHYMATOUS GOITRE

This variety, often known as simple or colloid goitre, probably includes two varieties, the endemic and sporadic, which are dependent to a certain extent upon the same etiological factors.

Etiology. Sex.—Women are more commonly affected than men. In sporadic cases the proportion may be as high as 20 to 1, but in endemic areas the ratio approaches equality. In the sporadic variety especially there appears to be a close relationship between the activity of the female generating organs and enlargement of the thyroid. In many normal women there is an enlargement of the gland during menstruation and pregnancy, and the same increase is often noticed in glands which are the seat of pathological enlargement.

Age.—The condition generally commences at the age of puberty, but endemic varieties may occur at any period of life. Occasionally it may be present at birth, several children of the same family being affected with the disease. This is more common in endemic areas.

Infection.—Evidence in favour of infection as a causative factor is steadily increasing. There are certain areas, such as Derbyshire, Switzerland, and parts of India, where goitre is very common. In the Gilgit Valley 20 per cent. of the people are sufferers, whilst in some parts of the canton of Berne 80 per cent. of the recruits are rejected for this disease. All these endemic areas are alike in that they are mountainous regions with a temperate or sub-tropical climate. It has long been known that

there was a relationship between the drinking-water and the presence of this disease, and that if the water supply was changed a rapid diminution in the number of cases took place. McCarrison has brought forward very strong evidence to show that there is no relationship between the geological structure of the soil and the presence of goitre, but that goitre can be produced both in man and in animals by drinking certain waters, the property which has this effect being destroyed if the water be sterilized by heat. The infective agent is carried to the water from the intestinal tract of those affected with the disease. Working on these lines, he was able to produce a simple goitre in himself and in other men. His evidence shows that the disease is not due to one specific infection but is a reaction to one or more different organisms. Farrant's work on the changes found in the thyroid with acute and chronic infections lends support to this view.

Pathology.—There is at first a diffuse enlargement of the gland, the normal outline being maintained. The capsule shows many dilated and thin-walled veins and the cut surface a large number of small vesicles filled with colloid material, these vesicles resembling in the early stages grains of sago. Later, as the vesicles enlarge, a honeycomb appearance is presented or definite cysts are seen. Hæmorrhage may also occur into the gland substance or into the cysts. In certain cases, in addition to the general enlargement, there are definitely encapsulated adenomata which are often richly cellular and therefore granular in appearance. In cases where there is clinical evidence of hyperthyroidism there is often but little colloid. The endemic varieties show, in the early stages, appearances identical with those of the sporadic cases. Cysts are, however, more common, the outline of the gland being irregular. The cysts themselves, owing to the breaking down of the intervening walls, become multilocular. In longstanding examples there may be a considerable overgrowth of fibrous tissue.

Microscopically, it may be seen, in addition, that the lining epithelium is often flattened, but may show a certain amount of proliferation in places.

Symptomatology.—In sporadic cases a swelling of the neck is first noticed, more commonly at the age of puberty. The swelling is discovered by accident. It increases at the menstrual periods or during pregnancy, and in

GOITRE

painless, smooth, and retains the outline of the normal thyroid gland. The swelling is attached to the trachea, moving on deglutition, and lies between the sterno-mastoid muscles. There may be a slight dragging sensation in the neck, and the superficial veins may be dilated. In about one-third of the cases there are no other symptoms, although the swelling may steadily increase in size and give rise to considerable inconvenience from this cause. In the remaining cases there is evidence of pressure upon the trachea, dyspnoea being present on exertion. Later there is difficulty in breathing in the recumbent position, and a stridor may even be present. This symptom is more prominent with fibrous and retrosternal goitres. Rarely, the oesophagus may be compressed, and occasionally pressure upon the trachea or recurrent laryngeal nerve may cause aphonia. Acute paroxysmal exacerbations of the dyspnoea may be present at night and may prove fatal. The endemic cases are more commonly irregular in outline and attack members of the same family. The offspring of such patients may be goitrous or cretinous. Parenchymatous goitres can be distinguished from the malignant varieties by the slowness of their growth and by their mobility.

Prognosis.—The sporadic varieties occurring in young girls tend to recover spontaneously, although they may persist for several years. The swelling may, however, increase in size and become cystic, fibrous, and irregular in outline. In such cases the immediate danger is the presence of dyspnoea, which, if not relieved, may be fatal. After months or years hyperthyroidism may appear and steadily progress to true Graves's disease. In other cases, especially if fibro-cystic, the normal gland substance may atrophy and the patient develop symptoms of myxoedema. In the endemic type the condition is liable to progress steadily unless the patient be removed from the infected area. Either type may become the seat of malignant disease.

Treatment.—In the early stages of the sporadic variety palliative treatment should alone be tried. Some form of iodide, either as an ointment or internally, is generally advocated. There is, however, a considerable danger of this provoking symptoms of hyperthyroidism, and for this reason neither iodide nor thyroid extract should be administered. In the early stages attention should be given to the general health, any focus of infection being sought for, especially in the intestinal

canal. For the latter, thymol in doses up to 10 gr. may be given, as advocated by McCarrison. In progressive cases X-rays may be tried, or inoculations with a vaccine prepared from the contents of the patient's intestinal canal, as used by McCarrison for the endemic varieties. In the latter the patient, whenever possible, should be removed from the infected area, and the above lines of treatment carried out. If the mass has persisted for several years, if it is sufficiently large to be unsightly, or if there is any evidence of pressure, especially on the trachea, operative treatment should be undertaken. In cases of acute dyspnoea operation must be performed. At times, relief may be given by division of the isthmus or removal of one lobe; but, occasionally, the trachea may be so collapsed that it is necessary in addition to perform tracheotomy. This operation should, however, be avoided if possible, lest sepsis of the thyroidectomy wound result.

MALIGNANT GOITRE

This condition is much less common than the simple variety, only about 1 in 30 or 40 of all cases being malignant. It may occur in a gland apparently previously healthy, or may be grafted upon one of the simple changes. In the former case the primary lesion is often minute, the attention only being directed to the condition by secondary deposits which are very prone to occur in the bones. It occurs, as a rule, later in life than the simple varieties, the most frequent ages of onset being between 30 and 50 years.

Pathology.—In primary cases only a small nodule of carcinomatous tissue may be seen, even when death has occurred from multiple secondary deposits. The nodule presents the aspect of a localized adenoma, but there is no apparent capsule. It appears solid, with little or no colloid, and is thus differentiated in the normal gland tissue. If the tumour has grown locally, the thyroid may be enlarged considerably and irregularly. It is hard, and in many cases has broken through the capsule of the gland to invade the surrounding tissues. It may have eroded the trachea or the overlying sternum. In secondary cases, carcinoma may be seen in only one area of the gland, but in this position may have invaded the surrounding structures. In all varieties, secondary deposits are common, especially in the bones. Microscopically the growth is richly cellular, and shows scattered throughout its

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substance small ill-defined alveoli filled with colloid material.

Symptoms.—There may be no symptoms in the thyroid gland, the secondary deposits being alone manifest. In other cases there is a rapid enlargement which shortly becomes adherent to the surrounding structures. There is early evidence of pressure and infiltration, the recurrent laryngeal nerve being frequently affected. The tumour is extremely vascular, large veins being seen on its surface, and the skin over it may become dusky and mottled. Pain is severe, dyspnoea and dysphagia appear early, and, after the growth has spread through the limits of the capsule, secondary deposits occur in the lymphatic glands. The growth may erode the oesophagus and the trachea, giving rise to hæmatemesis and hæmoptysis.

Prognosis.—Unfortunately, an accurate diagnosis is often not made until the growth has perforated the capsule and invaded the surrounding structures. If a diagnosis be made in the earlier stages, or if carcinoma be found after removal of an apparent benign swelling, the prognosis is better, but even in these cases deposits may appear in bones remote from the thyroid.

Treatment.—Operative treatment is alone of value. The whole gland should be removed, and myxœdema prevented by the administration of thyroid extract. If the growth is too advanced for removal, relief may be given by tracheotomy.

ALBERT J. WALTON.

GOITRE, EXOPHTHALMIC (see EXOPHTHALMIC GOITRE).

GONORRHEA.—An inflammation of the genito-urinary passages due to invasion by a specific micro-organism, the gonococcus. The infection may spread by continuity to adnexa of the genito-urinary passages, such as the prostate, vas, and epididymis in males, and the uterus, Fallopian tubes, and ovaries in females. It may also spread to other parts of the body by transference (a) on infected articles or (b) through the blood-stream. Wherever it occurs, gonococcal inflammation is at first purulent in type and tends to be followed by connective-tissue formation, which distorts or interferes with the function of the affected part.

Etiology.—Infection usually results from sexual intercourse, but may (very rarely in men, more often in women, and most frequently in female children) follow contact with infected

articles, such as closet seats, napkins, towels, etc. In the case of men sexual intercourse with an infected woman is followed by gonorrhœa in only a small proportion of cases (probably not more than 3 or 4 per cent.); it is the multitude of exposures which explains the great prevalence of the disease. In women infection is much more likely to follow impure intercourse, owing to the implantation of the virus in a protected situation.

Pathology.—The gonococcus, discovered by Neisser, is a diplococcus of which the two elements are kidney- or coffee-bean-shaped, with their concave, or flat, sides opposed (Plate 4, Fig. 5, Vol. I, facing p.146). In size it is about 1μ ,

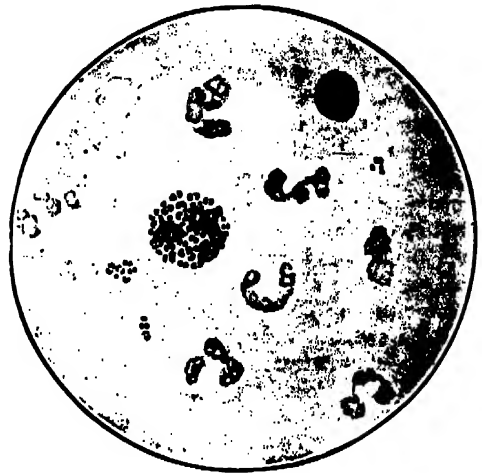


Fig. 30.—Pus in acute gonorrhœa. $\times 1,000$.
(Leedham-Green.)

or rather smaller than an ordinary staphylococcus. In films of gonorrhœal secretions it is found most characteristically within the pus-cells, in which it occurs in variable numbers; most of the cells of a gonorrhœal discharge contain none, some a few pairs, and a few may be found packed with gonococci, when the cell looks as if it had been peppered with shot (Fig. 30). For diagnostic purposes films should be stained by Gram's method, the gonococcus being distinguished from some morphologically similar organisms by failing to retain the violet stain which is first applied, and being then dyed by the brown or red usually employed as a counter-stain in Gram's method. The gonococcus cannot be distinguished either morphologically or by staining methods from two micro-organisms, *M. catarrhalis* and the meningococcus. Besides the fact that neither

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of these occurs usually in the genito-urinary passages, *M. catarrhalis* can be distinguished from the gonococcus by its ability to grow on plain culture media, the gonococcus requiring one which contains unheated constituents of blood. A fact of practical importance is the low vitality of the gonococcus, which will not stand complete drying, exposure to a temperature of 104° F. for six hours, or the application of comparatively feeble antiseptics.

When implanted on a susceptible mucous membrane, such as that of the urethra or the cervix uteri, the gonococcus quickly finds its way to the deeper tissues and invades the glands opening on the mucosa. Its toxin is irritant and erosive in action, and invasion is quickly followed by profuse shedding of epithelium, leaving large areas of submucous tissue bare; by free exudation of serum and polynuclear cells, constituting the purulent discharge which is such a prominent feature of the disease; and by infiltration of the deeper tissues with round cells, which may subsequently form scar tissue, with resulting constriction of the infected canal. The infection tends to spread along the mucous membrane, and far along canals opening on to it, giving rise to inflammation of structures so remote from the original site of infection as the vas deferens and epididymis in the male, the uterus, Fallopian tubes, and pelvic peritoneum in the female. The invasion of these structures is followed by inflammatory reaction similar in nature to that which occurs in the mucous membrane originally infected. Subsequently the deposition of connective tissue about the infected canal destroys its function; thus double epididymitis and double salpingitis result in sterility in male and female respectively. The inflammatory material imprisoned within an infected Fallopian tube may increase sufficiently to cause a large inflammatory cyst (pyo-salpinx) which may rupture into the peritoneal cavity. In the male, infection of the epididymis may not declare itself for many months, and then an attack of epididymitis may follow some trivial injury or strain. The pus-formation in epididymitis is rarely so great as to cause rupture of an abscess on to the external surface, the inflammatory material more usually being absorbed gradually, and the places where the inflammation was most intense—notably the tail of the epididymis—being occupied by one or more nodules of scar tissue, which can be detected by palpation long after the attack.

Much of the intractability of gonorrhoea is due to invasion of the numerous follicles which open on the mucous surface of the genito-urinary canal. Many of these become inflammatory cysts, with the infective material imprisoned within them safely out of reach of antiseptics applied to the surface. Invasion of the follicles gives the gonococcus the opportunity of setting up inflammatory reactions deep in the cavernous tissue, and the resulting deposit of connective tissue often causes distortion and narrowing of the urethral canal. Long after the mucous membrane has practically recovered, the contents of infected follicles may continue to be expelled at intervals on the surface, and give rise to fresh outbursts of inflammatory discharge. Sometimes the infective material bursts the confines of the gland, and an abscess forms in the surrounding tissues. This is seen in the periurethral abscess, prostatic abscess, and cowperitis of men, and the Bartholinian abscess of women.

It is probable that the gonococcus invades the blood-stream in most cases of gonorrhoea, but it is only in the minority—2 or 3 per cent.—that it gains a footing in other tissues such as joints, tendon-sheaths, bursae, the iris, and the endocardium, the first of these being most frequently affected, and the last by far the most rarely. The invasion of these structures is followed by profuse exudation of polynuclear cells and serum, and—a fact of some importance to the treatment—the production of new connective tissue in and about the affected part, which tends later to the formation of adhesions.

Symptomatology in men.—Both symptoms and complications are somewhat dissimilar in the two sexes on account of anatomical differences. In men, in an average attack, following the incubation period—which is usually about three days, but may be eight or ten or, very rarely, as long as forty—there is slight itching or irritation of the meatus, which appears redder than normal, with its lips stuck together by a slight mucoid discharge; examination of this shows epithelial cells covered with gonococci and a few pus-cells. This is the stage of the disease at which treatment ought to commence; if men would take alarm now and obtain skilled treatment at once, gonorrhoea would be a comparatively easy disease to treat, and the army of gonococcus-carriers would quickly vanish. But the average man either will not believe that it can be true that he, of all men, has caught gonorrhoea, or hesitates to

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seek advice because of the social stigma resulting from contraction of venereal disease. He waits a few days longer until the increase of symptoms convinces him, and by this time the golden opportunity of aborting the disease has been lost.

In the two days following the onset the symptoms increase rapidly; the discharge becomes creamy, or even bloodstained, and profuse; there is usually considerable burning and pain on micturition, and, at night especially, painful erections or chordee, in which the rigid penis is bent over in a curve, may become frequent. In a high proportion of cases the posterior urethra is invaded in the second or third week, and the backward spread of the disease may then be marked by great increase of painful symptoms, with frequency of micturition, strangury, and perhaps the passage of a few drops of blood at the end of micturition. It would be a mistake, however, to suppose that, without these symptoms, posterior urethritis does not exist, since in the majority of cases the infection of the posterior urethra is discovered only by objective examination. The discharge of purulent material into the urethral canal is shown also in the urine, which in the earlier stages is turbid with the pus that it has picked up in its passage through the urethra. Later, when the discharge has become so slight that none can be expressed from the meatus, it may be seen in the urine in the form of threads. On this account examination of the urine in gonorrhoea is a valuable diagnostic measure which will be referred to in more detail later.

In untreated cases the purulent discharge continues to be a prominent feature for six weeks or longer before beginning to die down. Most cases are treated before this, and usually by the end of the second or third week all discomfort has ended and the discharge become trivial. Favourable cases may be completely free from all signs in about six weeks, but, in very many, foci of infection remain which prolong the disease for months or even years. In these the discharge ("gleet") is discovered as a slight greyish bead in the mornings, or stickiness of the lips of the meatus, or simply as purulent threads or filaments in the urine. In some cases the prostate only may remain infected, and the sole evidence of disease consists in the presence of pus in the prostatic secretion. Often in these cases, owing to infection of the prostate, the patient complains of vague weight or soreness in the perineum and of various

neurasthenic symptoms. It is uncertain in what proportion of chronic cases of urethritis which started as gonorrhoea the gonococcus persists in the urethra. In most cases a secondary infection by other organisms, staphylococci, streptococci, and diphtheroids, occurs comparatively early in the disease, and it is these which are found in most of the old cases. Undoubtedly examples of persistence of gonococci for many years do occur, but they are in the minority, and in most cases no evidence of the presence of the gonococcus can be found by most careful search, either microscopical, cultural, or serological. The gonococcus is, however, notoriously difficult to cultivate from secretions containing mixed organisms, and the consequences of transference of infection to others are so serious that, in practice, it is safest to regard the patient as still suffering from gonorrhoea until all signs of urethritis have disappeared.

Local complications in men.—**Balanitis** from irritation by inflammatory secretions is fairly common wherever there is a long prepuce.

Periurethral infiltrate of some degree is so common in gonorrhoea that it might be accounted a routine feature of the disease, but in most cases the infiltrate is not easily detectable by palpation. In some cases a globular swelling forms somewhere along the urethral canal and may reach the size of a pea, a bean, or even a Tangerine orange. The abscess may burst externally or require opening; in most cases it gradually subsides to the size of a millet seed, and can be detected by palpation over a sound many months after the attack. These infiltrations are very apt to be followed by stricture unless care is taken to dilate the canal fully before the patient is dismissed. In very many cases a small residuum of infection remains within the follicle and is the cause of the attack persisting, or of repeated relapses. In the latter case all signs may disappear for weeks or months and then recur with a purulent discharge, which disappears again after a few days, the infected follicle having discharged its contents on to the surface and then closed down again.

Cowperitis is a comparatively rare complication. It is often diagnosed as prostatitis because of the accompanying pain in the anal region, pain on defecation, strangury, and even sometimes retention of urine. It is easily distinguished from prostatitis by palpating the acutely tender, swollen area between a finger

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within the anal orifice and a thumb just outside it. Usually the abscess bursts externally or is opened. Rarely it bursts into the membranous urethra.

Prostatitis in some degree is so common in gonorrhoea as to be reckoned a part of the ordinary disease, but that is the type which is only detected by the examination detailed below. In a small proportion of cases an abscess forms and gives rise to severe pain in the perineal region, pain on defecation, frequency of micturition, stranguy, and, often about the fourth day, retention of urine.

Examination per rectum reveals an intensely tender, swollen prostate. Relief is usually obtained by the abscess bursting into the deep urethra about the seventh day, when the urethral discharge, which has often almost ceased in the meantime, suddenly becomes enormously increased. The symptoms may be repeated a week or so later owing to regathering of the abscess if prostate massage has not been applied to keep the abscess draining into the urethra. Occasionally a prostatic abscess bursts into the rectum; very rarely indeed it may burrow and open in situations far removed from the prostate.

Vesiculitis rarely causes distinguishable symptoms. In cases of arthritis and other metastatic complications a chronic infection of one or both vesicles is often found; the affected vesicle is then easily palpable and tender.

Epididymitis is a relatively common complication, one testicle being affected in about 17 per cent. of cases, and both in about 1 per cent. It is a complication which is largely preventable and should not occur in more than 1 or 2 per cent. of cases. The chief causes are slight traumata such as may result from failure to reduce physical exercise and support the testicles properly, and undue irritation of the inflamed urethra by chemicals, instruments, or irrigation at too high a pressure. The attack usually commences with a smart rise of temperature and stoppage, or considerable reduction, of urethral discharge. There are often pain and tenderness at the inguinal rings, and if the right testicle is affected there may be pain in the right iliac fossa, which has led more than once to removal of the appendix. The inflamed epididymis becomes considerably swollen, and can be felt as a very tender bolster behind the testicle, which is also moderately inflamed. There is often also an inflammatory hydrocele, and the scrotal tissues are swollen and reddened. Very rarely an

abscess may form and burst externally. The acute phases of the attack usually last from ten days to a fortnight before the inflamed structures begin to show definite signs of subsiding, and one or more hard nodules may remain, particularly in the tail of the epididymis, for months or years afterwards. (See also EPIDIDYMITIS.)

Diagnosis in males.—In acute gonorrhoea the diagnosis is simple, since all that is necessary is to examine microscopically the discharge from the meatus and demonstrate gonococci in it. It is not so easy to determine in many cases how far the disease has progressed along the urethra, though Thompson's two-glass urine test is relied upon by many for this purpose. The patient passes a few ounces of early-morning urine into one glass and the balance into a second. If the first specimen contains pus and the second is normal, anterior urethritis is diagnosed; if both contain pus, anterior and posterior urethritis. The test will not detect a very early posterior urethritis, since not enough secretion has formed in the urethra to be forced into the bladder and mixed with the urine there, and the first urine often suffices to clear the posterior urethra as well as the anterior. A more certain method is to wash out the anterior urethra before letting the patient urinate. Then any pathological elements found in the urine must have come from the urethra behind the barrier formed by the compressor urethræ. In routine practice, in view of the time involved in washing the anterior urethra separately, it is safer and more expedient to treat all acute cases as if the whole urethra were involved, for then a very early posterior urethritis may be aborted.

In chronic cases, and where it is necessary to determine whether or not any infection remains, the task of diagnosis may be very difficult and complicated, and the examination is usually prolonged over a number of sittings. It is necessary to determine first whether or not the gonococcus is persisting, and secondly the sites of the residua. The steps of the examination are as follows:—

1. Wash the anterior urethra by irrigation with a good back-flow catheter until the washings are quite clear. The washings are examined microscopically. The first may be hazy and, on centrifugalization, show a deposit of pus in which organisms are found; or there may be threads of various types—long heavy ones which quickly sink and contain much

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pus; or light ones which are mostly mucus with a little pus and epithelium and indicate very slight trouble; or specks which, if few in number, are of no great significance. Pathological elements in these specimens indicate, of course, an anterior urethritis, and the nature of the micro-organisms its present cause.

2. Make the patient urinate into a series of glasses until the contents of one glass appear quite clear and free from threads. If then the patient still has urine in his bladder, let it remain; if not, irrigate the whole urethra with sterile tap-water or saline, leaving some in the bladder. The urine is examined on lines similar to those indicated above. It may contain long heavy threads of secretion from the surface, or (and) short comma-shaped ones which are plugs of secretion expressed from the mouths of the prostatic ducts.

3. Examine the prostate to determine if one or both lobes are unnaturally large, nodular, or boggy in consistence; massage the prostate, exercising gentle pressure from the periphery to the middle line. The last manœuvre usually causes prostatic secretion to appear at the meatus; this is caught and examined. The patient empties his bladder into a clean glass, and, if a good specimen has not been obtained at the meatus, the contents are centrifugized to obtain the deposit. Normal prostatic secretion is opalescent and contains round lecithin bodies, amyloid bodies rather like starch grains, epithelium, and a few leucocytes. Pathological secretion is much more turbid, sinks heavily to the bottom of the urine glass, and contains much pus, often in clumps. Again a close look-out is kept for micro-organisms.

4. Usually the seminal vesicles are examined at the same time as the prostate, and their contents expressed. Normal vesicles are difficult to palpate with a gloved finger, so that, if a vesicle can be felt easily, it is very suspicious of disease.

The examination so far has shown generally in what situations the disease persists—anterior urethra, posterior urethra, prostate, vesicles, or all of these. The further examination determines the nature of the disease.

5. The largest acorn-tipped bougie which will pass the meatus is passed down to the bulb. Normally it should not be stopped after passing the neck of the fossa navicularis until it reaches the triangular ligament. It may be stopped by an infiltrate or pass this with difficulty. The impression of an abnormality is confirmed by the patient's complaint of sore-

ness at that point, and by the fact that, when the shoulder of the bougie has been pushed past the obstruction, on pulling the instrument back it hitches against the obstruction.

6. A straight metal sound is passed into the urethra, which is palpated over it to detect the sites of abnormal swellings or thickensings.

7. A curved metal sound as large as will pass the anterior urethra is passed gently into the bladder. If it encounters obstruction in the posterior urethra or draws blood, persisting disease is indicated.

8. Urethroscopic examination is applied by far the more commonly to the anterior urethra, as posterior urethroscopy is usually attended by considerable discomfort to the patient. The conditions found are (a) general subacute inflammation, (b) soft and hard infiltrates which distort the normal contour of the urethra, (c) folliculitis, (d) granular patches or, particularly in the posterior urethra, definite polypi.

9. The gonococcal complement-fixation test in good hands often gives valuable indications in old-standing cases. Thus, if positive, it stimulates a further search for gonococci which may not have been found in the examinations detailed above; if negative and no gonococci have been found, it helps to confirm the impression that the gonococcus is not responsible for persistence of the symptoms.

10. After a specimen of blood has been taken for the complement-fixation test, and when the local disturbance consequent on the instrumental examination detailed above has died down, a large dose of vaccine, say 150 millions ordinary emulsion, or a dose of the special provocative vaccine made by Genatosan, Ltd., may be given. An increase of urethral symptoms following such an injection is suggestive of a persistent gonococcal infection.

11. It is advisable always to obtain specimens of urethral discharge on the day following any of the instrumental examinations detailed above, and examine them for gonococci. As additional stimulants may be mentioned the injection into the urethra of silver nitrate, 5-10 gr. to the ounce, or magnesium chloride, 1/500, and the consumption of alcohol, pickles, spices, and curries. After the use of any of these stimulants gonococci may be discovered in the resultant discharge.

12. Culture of the secretions stimulated by the procedures mentioned above is valuable, since on occasion gonococci may be discovered thus when microscopical examination has failed to detect them.

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The diagnosis of the various local complications is not usually difficult. Epididymitis may be due to other micro-organisms such as the tubercle or the colon bacillus. In tubercle the process is more chronic, the epididymis is more nodular, and there is much greater tendency to formation of multiple fistulae. Epididymitis due to *B. coli* is distinguished by the absence of any acute urethritis and the presence of *B. coli* cystitis.

Symptoms of gonorrhoea in women.—In women the disease is often so mild as to pass unnoticed by the patient. The chief sites of infection are the urethra, the ducts of Bartholin's glands, the vulva and vagina, and the cervix uteri, of which the last is by far the most frequent in adults. Acute urethritis gives rise to the same symptoms as in the male—urethral discharge and burning on micturition. The canal is a simple one, and in most cases the infection quickly clears up under treatment, but may persist in various follicles and ducts, notably Skene's ducts, which open in the floor just within the meatus.

Bartholinitis is comparatively uncommon. The infection may be confined to the duct, when the only sign is a reddened orifice from which pus can be expressed. In most cases the whole gland breaks down, with formation of an abscess. In chronic cases it is always necessary to inspect carefully the ducts of Bartholin's glands, since, without treatment, infection may persist here long after it has been cleared from other situations.

Gonorrhoeal vulvo-vaginitis is not a prominent feature of gonorrhoea in adults, but is the chief form in which the disease attacks female children infected by such external agencies as towels and napkins. There is a profuse discharge, with soreness and burning of the parts, which settles down in a few weeks to a chronic inflammation that may persist for months.

Cervicitis is much the commonest manifestation of gonorrhoea in adults, and may be the starting-point from which the disease spreads to the body of the uterus, tubes, ovaries, and pelvic peritoneum. A profuse discharge pours from the os uteri, which is usually reddened and excoriated. The patient may complain of no discomfort other than that due to the discharge, or there may be malaise with dragging pain and feeling of weight in the back and pelvic region. The discharge persists for months, probably owing to the depth to which the gonococcus can penetrate in the cervical glands and the difficulty of turning it out. It

is uncertain in what proportion of cases the infection spreads to the body of the uterus, but in many the intractability of the disease may be due to the endometrium having become infected, though there may have been no noticeable increase of symptoms. In a proportion of cases the upward extension of the disease is attended by a marked increase in pelvic and constitutional symptoms, which may confine the patient to bed for some days. In its chronic stages there may be constant back-ache and pelvic weight and menstrual disturbances. The uterus is found to be enlarged, boggy, and variably tender on palpation. It should be noted in connexion with subjective symptoms that their absence does not indicate absence of infection. It is the outstanding cases which influence book descriptions, and either much gonorrhoea of women is comparatively symptomless or the disease is not so prevalent in women as its known prevalence in men would lead us to believe.

Salpingitis, due to extension into one or both Fallopian tubes, results in partial or complete blockage of the tube, and is one of the commonest causes of ectopic gestation and of pyosalpinx. As an extension of the process, pelvic peritonitis may result from bursting of a pyosalpinx or from extrusion of the infected material through the fimbriated end of the tube. Similarly ovaritis may result from the entrance of gonococci through a ruptured Graafian follicle. The symptoms are severe constitutional disturbance, pelvic pain, and enlargement and tenderness of the affected structures.

Diagnosis of gonorrhoea in women.—Each of the parts likely to be affected is examined in turn, and, in taking specimens, it is necessary carefully to exclude contamination by secretion from other parts. The patient should not have urinated. After the vulva and vestibule have been disinfected the urethra is "milked" through the anterior vaginal wall and specimens made. At the same time the region within and around the orifice is carefully inspected for infected follicles. The urine may be passed at this stage and examined for pus and organisms. The orifices of Bartholin's glands are then inspected, and the glands squeezed to express any pus. The uterus, tubes, and ovaries are palpated bimanually for enlargement and tenderness, and a Fergusson's speculum is passed to expose the cervix. Specimens should be taken of vaginal secretion; this usually contains a great variety

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of organisms in which it is difficult to detect the gonococcus, but the examination is useful to show approximately the proportion of pus in the secretion which has collected in the vagina. It is advisable always to take cultures of the various secretions, since these may prove positive when no gonococci have been found in microscopical films. It should also be remembered that gonococci may be particularly difficult to find though subsequent events prove the case to be one of gonorrhœa, so that failure to find these organisms on one occasion is no criterion of cure; success is more likely just before or just after menstruation, and then after glycerin or an irritant such as silver nitrate, 10 gr. to the ounce, has been applied to the cervical canal.

The **complications common to both sexes** due to transference by infected articles are ophthalmia and proctitis, of which the former is dealt with in the article on CONJUNCTIVITIS. Gonorrhœal proctitis is more frequent in women owing to the greater opportunity of infection. It is accompanied by discharge from the anus and tenesmus. Later the cicatricial contraction resulting from the inflammatory deposits may lead to stenosis.

The chief **metastatic complications** requiring mention are arthritis, bursitis, tenosynovitis and iritis, which occur in about 2 per cent. of cases; endo- and pericarditis and pleurisy, which are extremely rare; and keratoderma. It should be remembered that in almost all cases prostatitis, or vesiculitis, or both are present.

Gonococcal arthritis may be mono- or poly-articular and may attack any joint, but the knee, ankle, wrist, tarsus, fingers, and elbow are most commonly affected in approximately this order of frequency, and arthritis of the temporo-maxillary joint is not uncommon. The affection may be acute from the first or begin very quietly. In the former case the affected joint presents all the usual signs of arthritis. Although in the acute stage the joint-fluid is purulent, there is no tendency to destruction of the joint as in septic arthritis. A feature of some diagnostic importance is that, though a series of joints may be affected one after the other, the inflammation does not leave any joint so completely as in rheumatic fever. The acute stage is commonly succeeded by a chronic, in which the affected joints are distended with fluid, or show the characters of a dry form of rheumatoid arthritis. Besides the limitation of movement due to pain, there

is always great danger of permanent stiffening from adhesions. (See also ARTHRITIS, SUPPURATIVE.) Bursitis and tenosynovitis commonly affect sheaths and bursæ around infected joints, though either may occur separately. Rarely a tendon-sheath may suppurate, but the main danger is the crippling from adhesions.

Iritis is commonly diagnosed as ophthalmia because of the reddened conjunctiva and the association with gonorrhœa. Here again the tendency to adhesions has to be guarded against.

Gonococcal endocarditis is a very fatal complication. The signs and symptoms of the variety recognized are those of malignant endocarditis.

Keratoderma blenorrhagica is a rare complication characterized by the formation of horny nut-brown nodules on the skin. The soles of the feet are most commonly affected, but the hands and in fact the whole body may be involved. Often it results in skin-casts of the whole soles of the feet and of the palms of the hands being thrown off. It is always associated with arthritis, which is often severe, and cases of keratoderma are notoriously intractable.

Treatment.—This is by no means satisfactory, as is testified by the multitude of remedies which are employed in it by different workers. It is only possible here to give a sketch of the main principles. The treatment is both general and local, the former being practically the same for both sexes. The **general treatment** aims at removing causes of irritation, assisting the process of elimination, and raising the patient's resistance. Generally, the first of these is effected by giving as much rest during the acute stage as is practicable and supporting such parts as the testicle. The urine is made as dilute as possible by giving large quantities of bland drinks, which also, by increasing the quantity of urine passed, aid elimination of the toxins lying within the urethral canal. Alcohol, spices, pickles, curried foods, and coffee are stopped, and the amount of meat is restricted. A smart purge is given at the outset, and thereafter the bowels are kept freely open by salines. A sedative diuretic mixture of potassium citrate, carbonate, or acetate, with belladonna, is prescribed. Some workers give sandalwood oil, cubebs, copaiba, or all of these, in the belief that they are specifics for gonorrhœa, but they are valuable only as sedatives, and are apt to upset the stomach and kidneys. Vaccines are

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valuable from the first, but do not act so effectively as in some other microbial infections. Those made of ordinary unheated emulsions of gonococci should be given in weekly doses, beginning with about 25 millions, and increasing gradually according to the reaction. Detoxicated vaccines, which give better results than ordinary emulsions, are administered in much bigger doses, commencing with 2,500 millions and increasing gradually in weekly doses to 10,000 millions.

Local treatment of acute gonorrhoea in males.—The local treatment may be divided into that which is suitable (a) for abortion of the disease, and is applicable only in the first two days, (b) for the well-developed acute stages, and (c) for the chronic stages. The abortive treatment which has given best results in my hands is to irrigate the whole urethra, as described below, twice daily for ten days, following each irrigation for the first five days with an injection of protosil 5 per cent., or similar organic preparation of silver. In the well-developed acute stage many preparations are employed for medication of the urethra. The most common are protargol 0.1-1.0 per cent., protosil 5 per cent., silver nitrate 1 in 10,000, acriflavine 1 in 5,000, and potassium permanganate 1 in 8,000-1 in 4,000. Of all these, none is better than potassium permanganate. The solution may be injected with a syringe of half-ounce capacity three or four times a day, but the method is inefficient from the cleansing point of view, and is attended by such disadvantages as the risk of spreading the infection backwards into the posterior urethra. It is a method which should be employed only when the patient cannot irrigate. The best results are undoubtedly obtained by irrigating the urethra twice daily with about two pints of solution. The irrigator used may be one made for the purpose, or an ordinary quart jug from which the solution is siphoned. It should be set at a height of about 3 ft. 6 in. above the penis when $\frac{3}{4}$ -in. rubber tubing is used. A bluntly conical nozzle should be employed, and this may be single or have a return-flow channel. After the anterior urethra has been distended a number of times with the solution, many workers stop if there is no sign of posterior urethritis. My own conviction is strongly in favour of allowing the solution to enter the bladder just as soon as the compressor urethra can be persuaded to relax. For this purpose the patient is told to breathe deeply and to

try to urinate into the nozzle, which is held firmly against the meatus, and presently a light thrill, communicated to the finger holding the penis, announces that the flow of solution, temporarily stopped when the anterior urethra was distended, has resumed. The operation is repeated about three times, the patient ejecting the solution each time he feels a strong inclination to empty his bladder. Under this treatment the attack may clear up completely in a few weeks, the irrigation being continued for about a week after signs have disappeared. A substantial proportion of cases require afterwards the treatment which is appropriate to chronic gonorrhoea. This is never instituted until the inflammation has very largely subsided and the urine is practically clear.

The treatment of chronic gonorrhoea in males is much more individual and intricate than that of acute. The measures employed depend largely on what is found as a result of the examination detailed under the heading of diagnosis. For *anterior* urethral residua, dilatation, with sounds and subsequently by means of a Kollmann's dilator, is mainly relied upon, but, through the urethroscope, diseased follicles can be destroyed by cauterization, and granular patches can be touched with fused caustic on a probe. *Posterior* urethritis is dealt with by passage of curved sounds, and the use of a Frank's or Kollmann's dilator about twice a week; by topical applications through the urethroscope as in *anterior* urethritis; and by instillations of silver nitrate 0.25-3 per cent., copper sulphate 0.2-2 per cent., or protargol 2-5 per cent., one of which is deposited drop by drop along the canal with an Ultzmann's or Guyon's syringe. Provided the resulting action subsides quickly and the patient shows steady improvement, instillation is practised twice weekly for a few weeks, the strength of the solution being gradually increased. Instillation may be useful in certain cases of superficial catarrh, but can be overdone, and then does more harm than good. The form of treatment most commonly employed in chronic gonorrhoea affecting the posterior urethra is massage of the prostate to empty infected follicles. By gentle pressure from the periphery towards the middle line each lobe is emptied in turn twice a week. Irrigation is continued throughout the treatment. The solution employed may be permanganate of potassium or of zinc, or the former with about $\frac{1}{2}$ gr. of zinc sulphate to the ounce. Commonly

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in these cases a mixed infection is found, and then mercury oxycyanide 1 in 4,000 is employed. Attention to the patient's general health is essential in the treatment of chronic gonorrhoea, and after two or three months of instrumental treatment, such as that detailed above, it is wise to give the patient a rest for two or three months, which he should spend as much as possible in the open air.

Treatment of local complications in the male.

—*Periurethral abscess*, if threatening to burst externally, should be aspirated and injected with electrargol, or may be incised. The same form of treatment is applicable to *abscess of Cowper's gland*. *Prostatic abscess* usually yields to rest in bed, mild purgatives, hot sitz baths, and an atropine suppository (gr. $\frac{1}{10}$) night and morning. Retention may have to be relieved by passage of a soft catheter for a few days. After the abscess has burst it is wise to massage the prostate gently every day so as to keep the opening into the urethra patent. *Epididymitis* may be cleared up very quickly by exposing the epididymis and lancing pockets of pus with a tenotome. A milder measure, which usually gives rapid relief, is to puncture the epididymis, running a fairly stout intramuscular needle along it from the cauda, and to aspirate by means of a syringe attached to the needle. Some workers inject about 1 c.c. of electrargol after the aspiration; it causes fairly sharp pain for some minutes, but the results are good. Soaking in water as hot as can be borne and the application of glycerin of belladonna are most useful. If the patient cannot rest in bed a well-fitting suspensory bandage, such as Jullien's, should be used.

Treatment of metastatic complications.—

Arthritis, *teno-synovitis*, and *bursitis* require rest at first with the application of sedatives. As soon as the patient can stand it, however, passive movements should be instituted to prevent adhesions. Ionization with chlorine or iodine ions, and radiant heat, are valuable. (See also ARTHRITIS, SUPPURATIVE.) In these, as in all metastatic complications, treatment of the deep urethra, prostate, and vesicles should never be neglected, since here is usually the laboratory from which the affected parts continue to receive fresh doses of virus. Gonococcal vaccines are valuable in all complications, as is also the therapy described variously as "pyrogenic" or "protein-shock." This consists in the injection of foreign proteins with the object of setting up a general reaction. A convenient agent to use is antityphoid vac-

cine, about 120 to 200 millions (contained in 1 c.c. of saline), which is injected intravenously every third or fourth day. Sterile milk, 5-10 c.c., injected into the gluteal muscles, gives similar results, as do also various substances which set up general febrile reaction when injected. *Iritis* requires hot fomentations or leeches to the temple, shading of the eye, and atropine drops to prevent the formation of synechiae.

In *keratoderma* measures are concentrated mainly on the treatment of the arthritis. In an extremely severe case of keratoderma in the Rochester Row Hospital a brilliant result followed a series of injections of novarsenobillon. The treatment was instituted on the suggestion of Prof. Bolam, who had experienced a similar effect in a very bad case of his own.

The local treatment of gonorrhoea in women depends on the part or parts affected. For *urethritis*, injections of protosil 5-15 per cent., protargol 1-5 per cent., or silver nitrate 0.25-1 per cent. are useful, but I prefer thorough irrigation of the canal with permanganate of potassium 1 in 8,000-1 in 4,000 once or twice daily. Residua of infection in Skene's ducts are dealt with by the application of 5-per-cent. silver-nitrate solution with a syringe through a blunt-ended needle. Infected follicles are destroyed by caustic or cautery through the urethroscope. Acute *bartholinitis* with abscess-formation usually requires a free incision. Chronic infection of the duct may yield to injections of strong silver nitrate or may require extirpation of the whole gland and duct.

Cervicitis is treated by different workers on a multitude of different plans, of which it is possible to mention only a few. Generally, all agree as to the necessity of a daily thorough cleansing of the vagina, which may be accomplished by douching (with potassium permanganate 1 in 8,000, mercury biniodide 1 in 2,000, or other antiseptic in suitable strength), or by swabbing with hydrogen peroxide, followed by careful drying out of the canal. Many workers content themselves with douching, but better results are obtained by following up the cleansing with applications to the cervical canal and vagina. Among the numerous applications may be mentioned painting the canal and vagina with 10-per-cent. silver nitrate, 50-per-cent. picric acid in glycerin, tincture of iodine in glycerin (1 in 8), tincture of iodine applied every third or fourth day. The application of iodized phenol (iodine 40 gr.,

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liquefied carbolic acid 1 oz.) to the cervical canal every fourteen days is favoured by many, and the insertion into the canal for six hours every third day of a strand of gauze soaked in glycerin of iodine (tincture of iodine 1, glycerin 8). After trying most of these applications with variable results, many of them disappointing, my present preference is for avoiding strongly caustic remedies in favour of a line of treatment which, while not increasing the damage already done by the gonococcus, and possibly damming back behind a slough the flow of serum from the deeper tissues, still promotes a free exudate and favours elimination of the deeply-lying gonococci. After thoroughly cleansing the vulva and vestibule, a Fergusson's speculum is inserted and the vaginal canal cleansed with peroxide of hydrogen until frothing ceases. Excess moisture is then removed by dry swabbing, and a Playfair's probe soaked in 10-per-cent. glycerin of ichthylol inserted into the cervical canal. This is renewed after a few minutes, and after removal of the probe the vagina is packed with gauze 2 in. broad, the first 9 in. of which is soaked in glycerin of ichthylol; this is paid off from a bandage-like roll through the speculum until the vagina is comfortably filled. The dressing is applied every day or every other day, the gauze being removed in any case after twenty-four hours.

In cases of *acute metritis* rest in bed is necessary, and hot douching, followed by insertion of glycerin of ichthylol tampons into the vagina. In *chronic metritis*, when other measures fail, curetting of the uterus, followed by painting with tincture of iodine, is sometimes recommended, but has a bad reputation for causing salpingitis. A milder measure is to dilate the cervix under general anaesthesia and paint the endometrium with picric glycerin or with tincture of iodine. Dr. R. Hobbs (Kensington Infirmary) has had excellent results from dilatation up to No. 7 Hegar, followed by syringing out of the uterus with weak iodine, and packing for six hours with a strand of gauze 1½ in. broad soaked in glycerin 8, tincture of iodine 1. This treatment is repeated monthly.

Vulvo-vaginitis of girls, due to accidental infection, is troublesome and difficult to treat on account of the smallness of the parts and the presence of the hymen. (See *Vulvo-vaginitis in Children*, under *VAGINITIS*.) Many workers now rely mainly on careful external cleanliness and vaccine treatment.

In children's wards the greatest care must

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always be taken to prevent the transmission of infection to other children. Similarly, mothers suffering from gonorrhoea should always be warned most carefully of the danger of infecting their own children with soiled fingers, towels, napkins, and even by contaminated bath water. L. W. HARRISON.

GONORRHEAL ARTHRITIS (see *GONORRHEA*).

GONORRHEAL OPHTHALMIA (see *CONJUNCTIVITIS*).

GOSSYPIOSIS (see *PNEUMONOCOINOSSES*).

GOUT.—A disease associated with imperfect metabolism of purin bodies, leading to the deposit of biurate of soda in the joints and elsewhere.

Etiology and incidence.—Gout is a rare disease except in England, Italy, and certain areas in Central and Northern Europe. According to Lindsay, the area of distribution is increasing with the tide of emigration from England, the home of gout. At the same time the frequency of gout—at any rate, in England—is diminishing. In part this diminution is only apparent, since more accurate methods of diagnosis have removed cases formerly considered gouty into other categories, but there is also a real diminution due to more temperate and careful methods of living, combined with physical exercise. For these reasons gout is more frequent in towns than in the country. As Sir Archibald Garrod says: "Gout has tended to become prevalent among peoples who, after periods of stress and struggle, have attained to conditions of material well-being, who have aggregated in cities, which become the centres of increasing luxury and more elaborate civilization." Heredity undoubtedly plays a part in the transmission of gout. It has been asserted that, like hæmophilia and pseudo-hypertrophic muscular paralysis, the disease, though attacking males, is transmitted more often by the female line. Lindsay, on the contrary, found the tendency to transmission to be four times greater along the male than the female side of the family.

Gout is decidedly commoner in men than in women. During a period of ten years, 114 men and only 11 women were admitted to the wards of St. Bartholomew's Hospital suffering from it. In women the disease most frequently occurs after the menopause, and it "more often smoulders without actually bursting into

flame." In Lindsay's series of cases the maximum incidence for males was between the years 30-34, declining after 44. This is earlier than has commonly been believed. His youngest case was a boy of 9. The greatest age at the time of the first attack was 68.

Occupation plays a very important part. Painters and other workers in lead are most liable to be affected. Occupations presenting facilities for indulgence in food and drink are also especially liable. Thus gout is common in butlers, cabmen, and workers in breweries. Sedentary habits are a contributory factor, more particularly in those who, in earlier life, were accustomed to take plenty of exercise.

Morbid anatomy.—Infiltration of the articular cartilage with crystals of biurate of soda is the characteristic lesion. This can be observed with the naked eye even in slight cases at a post-mortem examination. With the microscope it will be observed that this infiltration does not extend into the deepest layers of the cartilage nor actually to its surface. It is densest nearest to the surface, fading off gradually as it gets deeper. In early cases the deposit takes the form of acicular crystals which gradually form a dense felt-work. As the deposit accumulates the skin over the joint may become reddened and ultimately may ulcerate, giving vent to a yellowish semi-solid exudate of biurate crystals. With the X-rays clear areas in the bone can sometimes be seen. These are occupied by mucoid material containing uratic crystals which, of course, are not opaque to the rays. Apart from these, there is increased density of the bone; rarefaction of the cancellous tissue is not seen as in rheumatoid arthritis. The changes in the articular cartilages may result in partial dislocation, and all stages from fibrous to bony ankylosis are met with. Ironside Bruce called attention to deposits round the extremities of the phalanges which have been proved by Strangeways to be composed of true bone. Norman Moore found that the deposits chiefly occur in the metatarsophalangeal joints of the big toes, and then in the insteps, ankles, knees, hands, and wrists. The larger joints nearer the trunk are less frequently affected, though the temporomaxillary and sterno-clavicular joints may be involved, as in rheumatoid and gonococcal arthritis. Abarticular deposits are commonest in the ears, eyelids, nasal cartilages, and the bursæ over the olecranon and kneecap. Deposits may also be found in the lumbar and

palmar fascia and in the sheath of the sciatic nerve. They do not occur in the vital organs. Lindsay points out that the organs in which uratic deposit occurs are rich in sodium, while those, such as the liver and spleen, which are always free from deposits have a very low percentage of sodium.

It is clear that deposits, once formed, can be reabsorbed, as a joint which was known to have been the seat of gouty inflammation during life may fail to show any deposit after death. Apart from uratic deposits, the gouty subject is liable to show such changes as arterio-sclerosis and chronic interstitial nephritis with consequent hypertrophy of the heart. Emphysema is also common. Although the origin of the disease is probably hepatic, the liver does not show any constant morbid change.

Pathology.—Uric acid is tri-oxy-purin, $C_5H_4N_4O_3$, i.e. one of the more oxidized and less toxic members of the purin group which have, as Emil Fischer showed, the common nucleus C_5N_4 . The origin of uric acid in the body is partly from the food (exogenous) and partly from the tissues (endogenous). The exogenous sources of purins are—

1. Free purins, the xanthin and hypoxanthin of meat juices and extracts.
2. Bound purins, contained in the nuclei of cellular foods.
3. Methyl purins, the alkaloids of tea, coffee, and cocoa.

Milk, eggs, cheese, white bread, butter, rice, and most vegetables except the pulses are practically free from purins. Fish contains 4-8 gr. of purin nitrogen per lb. Ordinary meat contains 7-9 gr., though rumpsteak may contain as much as 15 gr. Chicken contains 9, liver 19, and sweetbread 70 gr. per lb. It will be noted, therefore, that the distinction usually drawn between red and white meat is entirely fallacious if it is intended, as it presumably is, to refer to the amount of purins they respectively contain, for chicken contains more than mutton, while sweetbread is exceptionally rich in them. Of the ingested purins a large part, varying from 50 to 90 per cent., is normally destroyed by the liver, being oxidized to uric acid and thence to urea; the excreted portion being that which has escaped destruction. This accords with Hopkins's observation that the principal increase in the output of uric acid occurs one hour after a meal, whereas the rise in urea does not take place till four hours later.

Endogenous purins amount to about 0.2 gm.

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of nitrogen a day; they appear to come mainly from the leucocytes and muscles. The enormously increased output of uric acid in leukaemia is associated with the great excess of leucocytes in the blood.

In the past it was assumed that the body could deal with its endogenous purins, and that toxic results were due to the exogenous purins. There is really no evidence of this, and, even on a strictly purin-free diet, a patient may continue to excrete cayenne-pepper crystals of uric acid. Two definite facts established by Sir Alfred Garrod still hold. In gout there is excess of uric acid in the blood and, before the paroxysm, there is a diminished output of uric acid in the urine. All this reveals deficient power in catabolizing purins. The unconsumed purins are not really poisonous any more than is the sugar not consumed by a glycosuric, but the resulting uric acid has the disadvantage of being but sparingly soluble, and is therefore apt to be precipitated in outlying parts of the circulation. Sir William Roberts held that the uric acid circulated as a quadriurate (H_2U , MHU), becoming precipitated on conversion into a biurate (MHU) by the abundant sodium salts in the synovial membrane. He showed that *in vitro* sodium salts prevented the resolution of a uratic deposit. It is probable, however, that the so-called quadriurate is merely a mixture of uric acid and biurate, and that uric acid normally circulates in an organic combination, possibly with thyminic acid. Diminished alkalinity of the blood probably does not play any important part in the deposition of biurates, though stagnation of flow and excess of sodium salts may well do so.

Another conception of gout, not inconsistent with the above, is that it is due to an intestinal toxæmia. Such a toxæmia might interfere with the hepatic functions in purin metabolism, just as cirrhosis of the liver results from the chronic gastritis of alcoholism or as phosphorus poisoning interferes with the fat metabolism of the liver.

Symptomatology.—In men the onset is usually sudden. Lindsay found it so in 85 per cent. of his male cases but in only 53 per cent. of the female. Often the patient has been feeling unusually well before an attack, though, sometimes, there may have been digestive troubles, or nervous symptoms such as neuralgia, cramps, depression, or marked irritability. The principal exciting causes are worry and, still more, indulgence in some article

of food or drink known to excite attacks. The patient has usually been asleep for a few hours and then is awakened by a severe pain in a joint, most usually the metatarso-phalangeal joint of the big toe. A damaged joint is more liable to attacks than any other, and this particular joint is frequently injured by the wearing of boots. The joint soon becomes hot, red, swollen, glazed, oedematous, and exquisitely tender. During the next day the pain diminishes, only to return in the night, though perhaps less severely. This may happen for several nights in succession, the pain becoming less as the swelling reaches its height. Usually the attack passes off after about a week, and the skin desquamates as the swelling subsides. During the paroxysm the temperature is moderately raised, and there are thirst and anorexia, and scanty, high-coloured urine.

There may be a considerable interval between the first and second attacks. Later, however, recurrences are apt to occur about once a year, and then at shorter intervals as more joints become involved. The attacks may be less severe but recovery is less complete, so that the joints, particularly those of the knuckles, become deformed, and "chalk-stones" may appear. Ultimately the skin over them becomes thin and red, and may give way. Under treatment the disease may be arrested or its severity may be diminished so that a progressive crippling of the joints may be avoided. A man may outlive the tendency to recurrence altogether, even without treatment. The deposit of tophi in the ear and eyelid is usually painless, but deposits in the fascia and bursæ may be acutely painful.

During the gouty paroxysm the urine is simply that of a febrile state, and, although the output of uric acid may be much diminished, it will have risen to normal by the second or third day of an attack, and may even exceed it—the so-called "uric-acid shower." It is often stated that attacks of gravel may alternate with the arthritic paroxysms. This is not common, however, nor would it be expected, considering that in the former the excretion of uric acid is abundant, while in the latter it is subnormal.

In chronic gout the urine is copious, of low specific gravity, nearly always containing less uric acid than normal and often a trace of albumin. The patient suffers from irregular twinges of pain in various parts, perhaps for weeks at a time. Lindsay calls attention to the bevelling down of the teeth which is so

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common in this state, though actual caries is not.

Irregular gout.—With more accurate methods of diagnosis, the number of the phenomena thus classed is diminishing. Hardly any characteristic of the middle and degenerative periods of life has escaped being thus designated by some authorities. Yet the proof that such phenomena have any real associations with gout is not forthcoming. The following symptoms are the most important that have thus been classified:

(1) *Skin affections.*—A dry, scaly eczema chiefly attacking the face, forehead, ear, and back of the neck is often called gouty, yet Adamson says that "a gouty patient is practically unknown in a skin clinic." There is no real evidence for regarding psoriasis as gouty.

(2) *Dupuytren's contraction* of the palmar fascia is often regarded as gouty, but is more probably infective in character, resembling fibrositis in this respect.

(3) *Special senses.*—Episcleritis and conjunctivitis (the "hot itching eye") and iritis may all certainly be met with in gouty patients. The evidence for a gouty origin of glaucoma is less satisfactory.

(4) *Respiratory system.*—Asthma may occur in members of a gouty family who do not themselves suffer from gout. No doubt many of the so-called examples of "asthma" in gout are really secondary to emphysema or implication of the heart or the kidneys, an entirely different condition from true bronchial asthma. The gouty are liable to emphysema.

(5) *Circulatory system.*—Chronic interstitial nephritis and atheroma may be set up by gout. They will produce changes in the cardio-vascular system which, rather than the gout itself, are responsible for symptoms. Recurrent phlebitis, especially in the legs, is often attributed to gout, and may be the cause of pulmonary embolism.

(6) *Digestive system.*—"Bilious attacks," with furred tongue, offensive breath, and constipation, are not at all uncommon, but it is just as likely that a person who has a defective hepatic metabolism will be unduly liable to *B. coli* infections of the liver.

(7) *Urinary system.*—So-called "gouty" cystitis and urethritis are really *B. coli* infections. The alleged association between gout and glycosuria is discussed in the article
DIABETES MELLITUS.

(8) *Nervous system.*—Migraine, neuralgia,

neuritis, and sciatica have all been attributed to gout, with varying degrees of probability.

Suppressed or retrocedent gout is a term applied to alarming and often fatal symptoms which are said to come on as a result of chilling of or the direct application of cold to the affected joints, the swelling of which may diminish. The direct association with cold is not always clear, and a consideration of the symptoms thus described makes it highly probable that they are essentially uræmic in nature. Thus, there may be symptoms referable to (1) the alimentary canal, such as pain, vomiting, and diarrhoea, (2) the cardio-vascular system, such as dyspnoea, palpitation, syncopal and anginal attacks, and (3) the nervous system, such as headache, twitchings, delirium, and coma.

Prognosis largely depends upon the amount of cardio-vascular change and chronic interstitial nephritis. If these are pronounced and the blood-pressure remains high, there is a liability to such accidents as heart failure, cerebral hæmorrhage, and uræmia. Apart from these complications, gouty patients tend to live long and may ultimately outgrow their tendency to attacks.

Diagnosis.—This has principally to be made from rheumatoid arthritis, osteo-arthritis, and the various forms of infective arthritis; less commonly confusion may arise between gout and tuberculosis of the joints or arthropathies such as a "Charcot's joint." The presence of tophi in the ears is a great aid to diagnosis. The small atavistic cartilaginous nodules known as "Woolner's marks" and little sebaceous cysts must not be confused with tophi. *Rheumatoid arthritis* is commoner in women than in men, the onset is usually more insidious, and the small joints of the hand are likely to be involved early. The effect of treatment will help to clear up the diagnosis, the gouty paroxysm yielding much more quickly. X-ray examination of the affected joint may be of assistance. In *osteo-arthritis* the onset is always insidious, and pain is not a prominent feature at first. The presence of grating or creaking in the joint, and of osteophytes, will help to decide the question, while an X-ray examination would probably be conclusive. A *pyæmic* or *gonorrhæal infection* is only likely to be mistaken for gout if it produces arthritis limited to a single joint. In the former case the *grave* constitutional state, the higher temperature, and the more rapid pulse should lead to a

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careful search for a septic focus. Gonorrhoeal infection leads to much more effusion in the joint itself, and is also liable to involve the surrounding tendons and fascia. Once suspicion is aroused, the diagnosis can be made by examination for pus or threads in the urine. *Tuberculosis* of the joints seldom causes confusion. The age and sex of the patient, a past or family history of tuberculosis, the much more gradual onset, and the absence of heat and redness will all help to settle the question. X-ray examination shows a characteristic foggy appearance of the bones in the neighbourhood of the joint which differs widely from the clear-cut outline seen in gout. *Charcot's joints* are painless and characterized by increased mobility. Routine examination of the nervous system for signs of tabes or syringomyelia would soon settle the question.

Treatment. (1) *The gouty paroxysm.*—The affected joint should be raised and protected. Cold applications or leeches should not be used. A hot lotion containing an ounce each of bicarbonate of soda, glycerin, and tincture of opium in 12 oz. of water usually affords some relief. Antiphlogistine applied hot is sometimes useful. It may be kept hot by a hot-water bottle. The most effective drug internally is colchicum. Its use is purely empirical; its alkaloid, colchicine, temporarily diminishes the number of leucocytes and is a stimulant to the intestinal muscles. Thus the endogenous formation of purins may be checked and absorption of toxins from the alimentary tract lessened, but we have no real proof of this. Ten minims of the vinum colchici or the tinctura colchici seminum should be given every four hours for a few days. It is usually combined with 10 gr. of carbonate of magnesia or salicylate of soda. The latter drug seems to relieve pain, though its use cannot be justified rationally, as it appears to increase the endogenous uric acid. Colchicum may cause purging and faintness in some patients, so that it must be used with caution; if it disagrees, phenoquin may be given instead on the lines described later. In any case, phenoquin is useful after colchicum. Citarin is a compound of formaldehyde and citric acid which is incompatible with alkalis. It has been recommended in doses of 15–30 gr. three times a day. Potassium citrate helps both by promoting diuresis and by rendering the urine less acid. A mixture of iodide and bromide of potassium must be resorted to if none of

the above drugs proves suitable. If the kidneys are sound, 10–15 gr. of Dover's powder may be given at night.

The diet should be light and such as is suitable for any febrile state. All meat juices and alcohol should be forbidden during the acute stage.

(2) *The gouty state.*—Some form of outdoor exercise is always advisable for the gouty, together with some occupation or hobby to prevent morbid introspection. Late hours and heavy dinners must be avoided. The bowels must be carefully regulated by an aperient water daily, reinforced once a week by the compound colocynth pill, or a pill containing 1 gr. of euonymin with 2 gr. each of iridin and extract of hyoscyamus.

The principal point in the regulation of the diet will be to diminish the intake of purins. Internal organs, such as liver, kidney, and sweetbread, are rich in purins, especially the last, and they should therefore be forbidden. Strong animal soups and meat extracts should also be forbidden, as they contain purin bodies without a corresponding amount of nourishment. Milk, eggs, cheese, white bread, butter, rice, and most vegetables except peas and beans are practically free from purins, and may, therefore, be allowed. Asparagus is usually forbidden, but the purin it contains is so little, only 1.5 gr. to the lb., as to be really negligible; the restriction therefore appears unreasonable. Oatmeal, on the other hand, contains 3.5 gr. of purin nitrogen per lb., and is not advisable. As far as vegetables are concerned, leaves are better than roots. Spinach may be allowed except when oxaluria is present. Fresh fruits, such as oranges, grapes, pears, and green figs, are quite suitable, though the sweeter, richer fruits, such as dates, are not. Lemons are sometimes forbidden because they are acid, but this is irrational, since the citric acid they contain will be converted into bicarbonates in the blood. Linnæus advocated strawberries for the gouty, but practical experience generally agrees with Garrod's objection to them. Garrod also advises that preserves should not be taken. Sugar is often forbidden, yet there does not seem to be any special reason for this except when there is a tendency to obesity. Fats have no real influence in gout except as a factor in obesity and as rendering food less digestible. When foods are cooked in fat they tend to diminish the secretion of gastric juice and thus cause dyspepsia, which is, no doubt,

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an exciting cause of an attack of gout by interfering with hepatic metabolism.

There can be little doubt that every gouty person is better without alcohol. Certain alcoholic drinks, such as port, champagne, liqueurs, and malt liquors, are especially liable to excite a paroxysm. Lighter white wines and well-diluted whisky are less likely to do so, and may be taken in strict moderation when the patient is unwilling to do without alcohol altogether, either because he is used to it or because he finds it an aid to digestion.

Tea and coffee are often forbidden on the ground that they contain methyl purins. These, however, are not converted into uric acid, but are excreted in the soluble form of xanthin and hypoxanthin. It therefore appears unnecessary to forbid them unless they cause indigestion. Cocoa, on the other hand, quite apart from its purin content, is not a suitable beverage, as it is too rich and fattening. It is not clear why hard water should be condemned, apart from its constipating effect. Presumably there is a subconscious but quite unjustifiable connexion in people's minds between hard water and "chalk-stones," which, of course, do not contain any calcium at all.

Mineral waters of all kinds have been widely recommended in the treatment of gout. So different, indeed, is the chemical composition of some of these that the sceptic may wonder how much of the benefit is attributable to the salts and how much to the water that contains them. Three classes of mineral waters have been specially used in this connexion: (1) *Radioactive waters*, such as those of Bath or Buxton, which may have some as yet undetermined influence on metabolism. (2) *Alkaline waters*, such as those of Vichy and Royat, which are presumed to aid in the solution of uratic deposits. (3) *Sulphur-containing waters*, such as those of Harrogate, Llandrindod, Strathpeffer, and Aix-les-Bains, which would appear to act as intestinal stimulants and disinfectants. The effect of drinking such waters may be reinforced by various hydrotherapeutic measures. Thus, if the skin has ulcerated over the chalk-stones, warm baths containing 2 per cent. of potassium carbonate will help to dissolve out the uratic deposit. Care must be taken not to leave the part in long enough to make the surrounding skin sodden. According to Llewellyn, hypothermal and sedative hydrotherapeutic measures are generally preferable to more stimulating pro-

cedures in chronic gout. He recommends a bath at 92° F., with the fan douche. Vichy or Aix-les-Bains douche-massage may be beneficial; the former raises blood-pressure and the latter lowers it, so that the blood-pressure should be observed to determine which to select for an individual case.

Hot-air or electric-light baths, followed by massage, may give good results when all acute symptoms have subsided. The Schnee four-cell bath is warmly recommended by some authorities. Ionization may be of decided assistance, lithium salts being used on the positive pole and 2 per cent. of salicylates or tincture of iodine on the negative. In the latter case a weaker current should be employed. A 1- to 2.5-per-cent. solution of sodium chloride may be used on the negative pole. A current of 5 ma. per sq. cm. of surface is passed for twenty minutes. The frequency of the application must be decided for each case.

Drugs have less influence in chronic gout than is usually supposed. Salicylic acid and phenoquin (phenylchinolin carbonic acid) are the only two which can be relied upon to increase the output of uric acid. Phenoquin does this in the case of a healthy person even on a purin-free diet, for about three days. Normally, an injection of uric acid increases the output for several days, but the total amount is not recovered. If phenoquin is given, the total quantity injected is excreted within twenty-four hours. Walker Hall finds the same result in a gouty individual, so that the drug would appear to cause the gouty renal cell to behave like the normal one. It may also shorten the acute stage of gout. These results might be claimed to support the retention theory of gout, but it is quite as likely that both retention and constitutional disturbances are produced by the same unknown perversion of metabolism. Phenoquin is prepared in tablets containing 7½ gr.; 4-6 of them should be given in the day, broken up in plenty of water. According to Weintraud, it is advisable to give sodium bicarbonate in full doses as well, ½ oz. on the first day and 1½ dr. on subsequent days. I have had good results by giving phenoquin for one week in each month in cases of chronic gout; Graham advises its administration on two consecutive days in each week. Owing to the largely increased output of uric acid which follows, it should never be used in cases of uric-acid gravel. It would appear more

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rational to give some at least of the alkali as a potash salt, since the sodium salts of uric acid are very insoluble. For a similar reason, Sir William Roberts advised the substitution of potassium chloride for table salt as far as possible. It is simpler not to use table salt at all.

Salicylic acid and its derivatives definitely increase the output of purins even after years of purin-free diet. This could not be due to washing out of retained purins which are normally destroyed rapidly by the liver. Another possibility is that the drug causes increased breakdown of nucleo-protein, thus setting free purin bodies, but in that case there would also be an increased output of phosphoric acid and a distinct loss of weight, neither of which results has been observed. The only remaining explanation is that salicylic acid produces synthetic formation of endogenous purins. In that case it is very difficult to justify the use of this drug in gout, and any empirical success only shows how little the metabolism of this disease is understood.

The evidence that other drugs increase uric-acid output is not satisfactory. Lithium salts have long been used for this purpose because lithium urate is much more soluble than the potassium or sodium salt, but chemical action is determined by the mass and avidity of the various interacting bodies, and also by the law that, in any mixture of acids and bases, it is the insoluble combination that occurs. A few grains of lithia introduced into the body cannot possibly contend with all the sodium present, which, in addition to having the advantages of mass reaction, forms the less soluble salt. Further, if enough lithium could be introduced for this purpose, it would have a toxic effect. Piperazine has a solvent effect on uric acid in the test-tube, but not in the body, because the presence of other salts inhibits its action. Lycetol and lysidin, closely allied substances, are open to the same objection. Hexamine was introduced as a uric-acid solvent because the formaldehyde to which it gives rise forms a soluble urate. It does not appear, however, definitely to increase

uric-acid output. Thyminic acid (soluro) was recommended by Minkowski in the belief that uric acid in the blood is normally kept in solution by combination with it. The drug, however, has had a very limited success.

Alkalis have been extensively used in the treatment of gout, though there is little experimental evidence in their support. Sir William Roberts finally decided against them, although, as he said, few had used them with more determination. It is noteworthy that, although there is often a definite diminution in the alkalinity of the blood in severe diabetes, this is not the type of glycosuria in which gouty symptoms occur.

Potassium iodide and guaiacum have also been used extensively. They may be given a trial, but my impression is that the cases in which the latter does most good are not really those of gout but of rheumatoid arthritis.

To sum up, in the treatment of chronic gout we have in the main to rely on hygienic and dietetic measures, reinforced by hydrotherapeutic and electrical applications to the affected joints. No doubt in the future, when more is known of the cause of the hepatic insufficiency, treatment may be based on more rational lines, especially if an intestinal toxin can be proved to be responsible.

W. LANGDON BROWN.

GOUTY KIDNEY (*see* NEPHRITIS).

GRAND MAL (*see* EPILEPSY).

GRANULAR KIDNEY (*see* NEPHRITIS).

GRANULOMA, INGUINAL (*see* ULCERATING GRANULOMA OF THE PUDENDA).

GRANULOSIS RUBRA NASI (*see* SWEAT-GLANDS, AFFECTIONS OF).

GRAVEL.—The presence of gritty particles in urine. (*See* URINE, EXAMINATION OF).

GRAVES'S DISEASE (*see* EXOPHTHALMIC GOITRE).

GUMS, AFFECTIONS OF (*see* STOMATITIS AND GLOSSITIS).

HABIT, DRUG (*see* DRUG HABIT).

HABIT SPASMS (*see* TICS AND HABIT SPASMS).

HÆMAGGLUTINATION (*see* SEROLOGICAL DIAGNOSIS).

HÆMARTHROSIS.—Hæmarthrosis is the result of bleeding into a joint, and may be due to several conditions, of which the most important are hæmophilia, scurvy, sprain, and fracture.

Pathology.—In traumatic cases the blood is eventually absorbed, but it often causes fibrous adhesions. In hæmophilia late changes take place at the edges of the articular surfaces and lead to lipping and loss of movement.

Symptomatology.—In hæmophilia there is often no history of injury, but one of the larger joints, especially the knee, shoulder, or elbow, is suddenly found to be swollen; there are slight heat and tenderness, and the joint is in the position of greatest capacity; there is little pain; the signs and symptoms are less acute than in infective arthritis, nor does their intensity increase as time elapses. On palpation there is a typical sensation of "bogginess," and the general temperature rises at night to about 100° F. In the late stage, when bone changes have taken place, there is limitation of movement and much grating; but there is no outgrowth of bone beyond the limits of the articular surfaces, as there is in osteo-arthritis.

Diagnosis.—It is most important not to mistake a hæmophilic joint for *infective arthritis*; even simple needling might lead to the death of the patient. A history of previous hæmorrhages, the presence of bruises elsewhere, deformities of other joints, and the family history will establish the diagnosis.

The late stage may be distinguished from osteo-arthritis by the history and by the fact that bony changes are entirely within the attachment of the capsule.

When there is a fracture, with a crack running into the joint, the nature of the swelling is unlikely to be mistaken.

Treatment.—In the traumatic form, particularly in the knee, aspiration on the third day hastens recovery and prevents adhesions, but must on no account be resorted to if circumstances prevent perfect asepsis. A firm bandage is then applied over a thick layer of wool,

and the joint splinted. Early massage is employed, and active movements as soon as most of the swelling has subsided.

In the hæmophilic variety the joint is immobilized and an ice-bag applied; morphia is given by mouth to relieve pain and restlessness. Treatment is also directed to the general blood condition, half a pint of horse-serum being given per rectum, and repeated within ten days to avoid anaphylaxis. Blood-transfusion is of great value if means are available for carrying out the special technique.

In the late stage, with bone changes and limitation of movement, massage and radiant heat help to increase mobility.

C. W. GORDON BRYAN.

HÆMATEMESIS (*syn.* Gastrorrhagia).

Etiology. Local causes.—The commonest sources of large amounts of vomited blood are peptic ulcer, duodenal ulcer, and œsophageal hæmorrhoids, resulting from the chronic passive congestion of the portal circulation. This last variety is most commonly produced by cirrhosis of the liver, but may arise from right-heart failure. Gastrostaxis, or gastric oozing, is an occasional cause of profuse hæmorrhage, especially in young women. In such cases there is no ulceration recognizable by the unaided eye, but the severity of the bleeding has often led to surgical interference on the erroneous assumption that ulceration is present. Another variety of hæmatemesis which may lead to confusion in diagnosis is that which is due to engorgement of the gastric capillaries in consequence of inflammatory diseases of the bowel, more particularly appendicitis. Carcinoma of the stomach may cause severe hæmatemesis, but more commonly the amount of blood is not large, and forms only a small part of the vomited material; it is then dark in colour, giving the appearance of "coffee grounds." Aneurysm of the aorta, by leaking into the œsophagus, may cause small hæmorrhages, or by rupture may produce one which is profuse and rapidly fatal. Serious hæmatemesis may also be due to venous congestion associated with enlargement of the spleen, especially in Banti's disease and thrombosis of the splenic vein. It is an occasional complication of abdominal operations. Mechanical injury and corrosive poisoning are easily ascertainable causes. A

rare local cause of considerable and even fatal hæmatemesis is congenital pyloric stenosis. Moderate or trifling bleeding may be due to slight degrees of congestion, whether active as in gastritis, or passive as in heart disease.

General causes. *Toxic.*—Among the general toxic causes must be included poisons such as arsenic and phosphorus, and the toxins of diseases: acute yellow atrophy, infective and toxæmic jaundice, smallpox, yellow fever, malaria, and the hæmorrhagic forms of the exanthems.

Blood diseases.—Pernicious anæmia, leucæmia, hæmophilia, purpura, scurvy, and hæmorrhagic disease of the newly born must be included.

Urgent vomiting from any cause may lead to slight degrees of hæmatemesis, the blood appearing either as bright-red streaks or as coffee grounds. It is thus found in such different conditions as sea-sickness, cyclical vomiting, postanæsthetic vomiting, whooping-cough, the pernicious vomiting of pregnancy, and acute intestinal obstruction.

It must not be forgotten that vomited blood may not originate in the stomach, but may have been swallowed or have arisen in the œsophagus. The bleeding in such cases may be an epistaxis, or the blood may come from a bitten tongue in epilepsy, or from the gums. It is unnecessary to enumerate all the causes concerned, but of some importance in diagnosis are the hæmatemesis which follows hæmoptysis and that which follows gum-sucking. The variety seen in hysteria and in mental disease is probably to be explained by this practice when no more obvious self-inflicted injury is indulged in. Hæmatemesis in nurslings is sometimes explained by the swallowing of blood from a cracked nipple.

Diagnosis.—In distinguishing between hæmatemesis and hæmoptysis a history alone is often very misleading, and even the most detailed questioning may fail to bring conviction. In hæmatemesis the blood is brought up by vomiting which is preceded by giddiness and faintness, it is dark in colour, clotted or grumous, often mixed with food and acid in reaction. In hæmoptysis it is coughed up, preceded by a tickling in the throat, frothy, bright in colour, contains only small clots, and is alkaline. Moreover, after the attack the sputum may be blood-tinged for several days. Evidence of a sufficient cause such as gastric ulcer or cirrhosis of the liver in the case of hæmatemesis, and of phthisis in that

of hæmoptysis, may establish the diagnosis. Though mælena may follow hæmoptysis, it is more prominent after hæmatemesis.

The possibility of the blood having been swallowed, or of deception on the part of hysterical patients, must always be borne in mind.

Treatment.—The treatment includes that of its cause. In small hæmorrhages this is all that is necessary, but in those which are severe more immediate measures are indicated. The patient should be laid horizontally and kept quite still, and an ice-bag may be applied to the epigastrium. No food or stimulants should be given by the mouth, but ice or iced water is permissible. Morphia $\frac{1}{4}$ gr. should be injected subcutaneously. Styptics by the mouth are less valuable; perhaps the best is adrenalin hydrochloride (1–1,000) in 20- to 30-min. doses. Normal horse-serum is advocated. To counteract the loss of blood, saline infusions may be required. Blood transfusion has recently been employed with considerable advantage.

FREDERICK LANGMEAD.

HÆMATOCELE.—An effusion of blood into the tunica vaginalis.

Etiology.—The effusion is probably always the result of some injury, though this may be trivial. A hydrocele (q.v.) is often already present, and the hæmatocele may develop after tapping, owing to the accidental puncture of a vessel; or it may be due to the rupture of a vein as the result of the sudden loss of support. Injury may also cause a hæmatocele apart from pre-existing hydrocele, or lead to hæmorrhage into a hydrocele apart from treatment. The rare apparently spontaneous cases are probably due to some trivial injury. Occasionally the onset is gradual, suggesting repeated small hæmorrhages, and the apparent non-traumatic origin may depend upon some pathological condition of the vessels. Lastly, hæmatocele may occur with malignant growths of the testis.

Pathology.—The testicle is generally situated at the lower and posterior part of the swelling. In recent cases the effused blood is partly fluid and partly clotted; in old cases there is usually thick, dark, altered blood, often containing crystals of cholesterin, and firm, dark clot which may be laminated. In these old cases the sac becomes very thick owing to the organization of layers of clot, and it may be of cartilaginous consistence, or even calcareous. Associated with these changes the testicle undergoes fatty or fibrous

HÆMATOMA

degeneration. Suppuration may occur in either early or late cases.

Symptoms and diagnosis.—Typically, there is rapid development of a scrotal swelling following injury and accompanied by pain and possibly by ecchymosis of the scrotum. The tumour is heavy, not translucent, and, though it may be entirely fluid at first, usually shows signs of fluid in places and appears to be solid elsewhere. The diagnosis may be verified by drawing off blood with a needle or a trocar and cannula. Hæmatocele may have to be diagnosed from hydrocele and from scrotal hernia, and it may be difficult or impossible to distinguish between a slowly developing hæmatocele and a malignant growth of the testis.

Treatment.—In recent cases the patient should be kept in bed with the scrotum raised, and cold applied in the form of either an ice-bag or an evaporating lotion. Should this not be successful an incision should be made and all blood and clots turned out. Drainage will be required for a few days, and every precaution must be taken to avoid infection. When a hydrocele is present an operation for radical cure should be performed by excising the parietal part of the tunica vaginalis. Care is required to avoid injury to the vas, which may be obscured by the effusion. Should a hæmatocele become infected, free incisions and drainage are necessary. In old-standing cases with thickening of the sac, excision of the testicle with the whole tunica vaginalis is the best treatment, since the testicle is almost certainly atrophied and functionless, and simple removal of the parietal portion of the thickened sac would most likely be followed by further troublesome oozing.

PHILIP TURNER.

HÆMATOCELE, PELVIC (see PREGNANCY, EXTRA-UTERINE).

HÆMATOCOLPOS (see AMENORRHOEA).

HÆMATOMA.—Like a bruise, hæmatoma is due to blunt force, but the blood, instead of being disseminated throughout the tissue spaces, is collected in a large single cavity of laceration.

The **symptoms** are the same as those met with in contusion (see BRUISES), with the addition that there is a definite fluctuating tumour to be felt. When the blood coagulates, a soft crepitus is to be discerned in place of fluctuation. The clot so formed is gradually absorbed, leaving in its place fibrous tissue.

HÆMATOMYELIA

Sometimes the clot shrinks and forms a peripheral layer surrounding the centrally-placed expressed serum, and absorption may fail to take place. Organization of the wall of clot follows, and the result is a serous cyst. These cysts vary greatly in size. They may be quite large, for example, in the thigh, where they form rather flaccid, painless, fluctuating swellings of which the precise boundaries are difficult to determine. A hæmatoma of the scalp, with its hard periphery of clot and central softened area, is liable to be taken for a *depressed fracture of the skull*. The similarity arises from the fact that as the clot of the hæmatoma shrinks it withdraws to the periphery, exuding serum into its centre. The margin is quite hard, and is apt to feel like the margin of a depressed fracture. A correct diagnosis is made by noting that this margin is elevated above the general level of the surrounding skull. Also, a hæmatoma may closely resemble an *abscess*, especially as the absorption of blood-clot is often accompanied by a rise of temperature. It must not be forgotten, however, that a hæmatoma may itself become infected and suppurate.

The **treatment** is similar to that of bruises. A persistent cyst may be treated by aspiration and the injection of 3 or 4 min. of liquid phenol, or, better, may be excised. When, from the position or size of the cyst, excision is impracticable, when it is too big for the injection of a few minims of carbolic acid to hold out any prospect of success, or when, excision having been excluded, aspiration has failed, the cyst should be incised, and its cavity packed with ribbon gauze. Gradually it becomes obliterated by the growth of granulation tissue. A suppurating hæmatoma is to be treated as an abscess.

C. A. PANNETT.

HÆMATOMA VULVÆ (see VULVA, DISEASES OF).

HÆMATOMETRA (see AMENORRHOEA).

HÆMATOMYELIA, or hæmorrhage into the spinal cord, is a rare condition apart from gross traumatic injury of the vertebral column. Even when the spine is fractured, dislocated, or severely concussed, there are usually small punctiform extravasations of blood rather than large hæmorrhages.

Spontaneous hæmorrhages are seldom due to arterial disease or a high blood-pressure alone; in most cases they result from the rupture of a

HÆMOCHROMATOSIS

vessel into a tumour, or into a syringomyelic cavity. The symptoms are sudden loss of power in the limbs with disturbances of sensation. The paralysis and anaesthesia diminish gradually, and leave a stationary condition resembling syringomyelia. As the blood destroys the grey more easily than the white matter, local wastings of muscles are generally a prominent feature. (See SPINAL CORD, LOCAL LESIONS OF).

GORDON HOLMES.

HÆMATOPORPHYRINURIA (see URINE, EXAMINATION OF).

HÆMATOSALPINX (see AMENORRHOEA).

HÆMATOTHORAX (see HÆMOTHORAX).

HÆMATURIA (see URINE, EXAMINATION OF).

HÆMOCHROMATOSIS.—A disorder of metabolism characterized by the presence of a large quantity of an iron-containing pigment in the internal organs, especially the lymphatic glands, the liver and the pancreas, by fibrosis of the liver and pancreas, and subsequently by bronzing of the skin. The symptoms are those of hepatic cirrhosis or, later, of pancreatic diabetes (bronzed diabetes).

Etiology.—The male sex is almost exclusively affected. Abbott's well-known patient "blue Mary" died from hæmatemesis due to cirrhosis before the onset of diabetes. Roberts's case appears to be the only example of bronzed diabetes in a female. The disease occurs mainly between the ages of 40 and 50.

Pathology.—At one time it was supposed that chronic hæmolysis, possibly due to infection with *B. coli*, gave rise to infiltration of the tissues with hæmosiderin, and that subsequently fibrosis occurred in the liver and pancreas. But as there is no evidence of anaemia, hæmolysis, hæmolytins in the blood, or of hyperplasia of the bone-marrow, and as the distribution of the pigment differs from that in pernicious anaemia, it is probable that a metabolic change, associated with visceral fibrosis, leads to retention and fixation of iron in the body. It has been thought that the hepatic cirrhosis is primary, and the pigmentation an exaggeration of that often seen in this disease. The cells and fibrous tissue of the liver, pancreas, secreting and lymphatic glands, especially those of the retroperitoneal space, contain the ferruginous pigment hæmosiderin, whereas an apparently iron-free pig-

ment, "hæmofuscin," has been described in the heart and muscular coat of the intestines. But, possibly, hæmofuscin is a compound containing iron in a more intimate state of combination than hæmosiderin. The liver is enlarged, shows multilobular cirrhosis, is deep red or maroon in colour, less pervious than in health to X-rays, and gives Perles's test (potassium ferrocyanide and hydrochloric acid) for iron. The hepatic artery shows endarteritis obliterans. The spleen is enlarged, firm, and pigmented. The pancreas is enlarged, pigmented, and shows fibrosis which may be so advanced as to cause diabetes. The intestines are pigmented.

Symptomatology may be that of hepatic cirrhosis with gastro-intestinal hæmorrhages and ascites, or later of diabetes. The liver and spleen are usually enlarged. The cutaneous pigmentation comes on later, and chiefly occurs on the exposed parts of the body.

Diagnosis depends on the association of cutaneous pigmentation with cirrhosis or diabetes. The pigmentation must be distinguished by the history from that of malarial melanæmia, and from that of argyria due to the medicinal use of silver and now seldom seen; from that sometimes present in generalized melanosis by examination of the urine for melanogen. In the pigmentation of ochronosis due to alkaptonuria the urine reduces Fehling's reagent but does not contain sugar, and in cases of ochronosis due to chronic carboic-acid poisoning it does not reduce Fehling. In some cases of splenic anaemia the skin is pigmented, but the spleen is much larger than in hæmochromatosis. From Addison's disease hæmochromatosis should be distinguished by the glycosuria and the enlarged liver and spleen.

Prognosis.—Life may be prolonged for years, and then death may occur from hæmatemesis or ascites. The onset of diabetes makes the prognosis very grave, as the fatal termination is seldom delayed beyond a year.

The **treatment** is that of hepatic cirrhosis (see LIVER, CIRRHOSIS OF) or of diabetes mellitus (q.v.).

H. D. ROLLESTON.

HÆMOGLOBINURIA (see URINE, EXAMINATION OF).

HÆMOGLOBINURIO FEVER (see BLACK-WATER FEVER).

HÆMOLYTIC TESTS (see SEROLOGICAL DIAGNOSIS).

HÆMOPERICARDIUM.—Pure blood is rarely found in the pericardium. Rupture of an aneurysm of the first part of the aorta is an occasional cause. If the aneurysm be in the intrapericardial part of the aorta, rupture may occur when it is of quite small size, perhaps no larger than a hazel-nut. Other causes are aneurysm of the coronary arteries and of the cardiac wall, and rupture and wounds of the heart. Death usually follows rapidly with symptoms of heart failure, but cases have been reported in which patients have lived for days with symptoms of heart failure and signs of pericardial effusion after cardiac rupture.

The inflammatory exudate is sometimes hæmorrhagic in pericarditis, especially that due to virulent rheumatism and to tubercle, Bright's disease, and new growth.

FREDERICK LANGMEAD.

HÆMOPHILIA.—A disease characterized by a chronic liability to immoderate or uncontrollable hæmorrhages, often from such trivial injuries that they may appear to be spontaneous. It is hereditary, and, though transmitted exclusively by females, is seen in males only.

Etiology.—Most of the cases have been recorded in Germany, Great Britain, or North America, the Latin races providing very few examples and the tropical countries practically none. Though decidedly rare, the disease is so striking that most cases are reported, and Bulloch and Fildes were able to analyse 946 papers and to reproduce 235 pedigrees in a monograph which has done much to consolidate our knowledge on hæmophilia. It has thus been shown that women, although they alone transmit the disease, are never true "bleeders." This interrupted or sex-limited descent is also seen in partial albinism, in which the eyes are chiefly affected, and in colour- and night-blindness. Hæmophilia is probably the most inheritable of all disorders. Bulloch and Fildes find very little evidence to support A. E. Wright's view that the disease may arise *de novo* from the accidental conjunction of two persons whose blood picture is that described by him as characteristic of hæmophilia. The women of "bleeder" stocks are exceptionally prolific, and an unusually high proportion of their offspring are males. In about 70 per cent. of the cases the disease appears before the end of the second year.

Pathology.—The anatomical view that hæmophilia depends on abnormal thinness

(hypoplasia) and fragility of the vascular system can no longer be maintained, and it is generally agreed that the essential factor is some defect in the coagulability of the blood, the exact nature of which has given rise to much debate. As they have a practical bearing on treatment, the following changes in the blood, discussed by A. E. Wright, may be mentioned, viz.: (1) deficiency in calcium, (2) leucopenia, (3) lack of fibrinoplastic substances (thrombokinase) in the tissue fluids, which mix with the blood as it escapes from the vessels, and (4) diminished CO₂-content, an increased CO₂-content being associated with increased coagulability of the blood.

The blood shows some leucopenia and a relative diminution in the polymorphonuclear leucocytes. The blood-platelets are not diminished. The coagulation time of the blood is generally prolonged—even to 60 minutes or more—but even here there is some divergence of opinion, and remarkably different results have been reported. The arterial blood-pressure is low.

Symptomatology.—Injuries so slight as to pass unnoticed by an ordinary person may give rise to bleeding which, persisting as an oozing in spite of pressure and other forms of treatment, may cause death or—and this is more usual—stop spontaneously when the patient is profoundly anæmic. The blood may then become almost colourless, the pulse quickens, cardiac murmurs, restlessness and excitability, thirst and headache appear. The blood, which fails to form a clot in the leaking vessels, coagulates as it drips from the oozing surface, and may form an enormous tumour. After cessation of the bleeding the red blood-corpuscles soon regain their normal number, but the colour index remains low for weeks or even months. The hæmorrhages may be external or internal. Epistaxis is very common. Circumcision may reveal the existence of the condition, and extraction of a tooth has often proved fatal; but blood may be drawn from a finger-prick or from a vein without danger. Hæmorrhages small or large may occur into the skin, and less often from the alimentary and urinary tracts. Severe bleeding from the umbilicus in the newborn is usually due to infection, and is very seldom a manifestation of hæmophilia. Bleeding into the peritoneum and pleuræ is most exceptional. Wright insists on the special nocturnal incidence of the hæmorrhages. Articular hæmorrhages are very characteristic, and occur in nearly all the

HÆMOPHILIA

cases; the onset is usually between the fourth and seventh years, and the liability to attack diminishes with advancing years. The knees are the commonest site—in about half the cases; next the elbow, in about 25 per cent.; and then the ankle and other articulations. Several joints may be affected in the same patient. Three stages have been described. In the first the joint is rapidly distended with blood, and is painful but not tender, the overlying skin is stretched but not red or hot. Absorption may follow, so that the joint is little the worse. In the second stage a form of chronic arthritis has resulted from repeated attacks; the synovial membrane is thickened and rusty coloured, and the cartilages degenerate. The clinical resemblance to tuberculosis is very close. In the third stage the articulations become permanently deformed, partially ankylosed, and show osteo-arthritic changes.

Although the liability to hæmorrhage is chronic, its intensity varies in the same patient, and may do so in a remarkable fashion. Prodromal symptoms, such as headache, hysterical restlessness, lassitude, and slight puffiness of the face, have been described. There may be extensive dental caries, which may be correlated with a deficiency of calcium, as shown by an abnormal appetite for chalk and dirt. A curious feature about some "bleeders" is concealment of their infirmity even when specially questioned before a proposed operation. A boy in St. George's Hospital asserted almost with his last breath the *idée fixe* that he did not bleed more than others (Dent).

Prognosis.—Age has a very definite influence on the outlook, which, on the whole, is bad in early life, for 50 per cent. of the patients die before the eighth year, and many more before puberty. The first hæmorrhage, however, is seldom fatal. The prognosis, which naturally depends on the severity of the symptoms, therefore improves as the patient gets older.

Diagnosis.—The essential features are the chronic liability to hæmorrhages in a male with a family history of the same disease. Multiple hereditary telangiectases may fulfil these requirements, but the presence of angiomas should prevent any mistake. The long course and the heredity should distinguish hæmophilia from hæmorrhagic states such as chronic purpura and acute leucæmia, which would present their special characters. In *purpura* the blood-platelets are diminished,

whereas in hæmophilia they are normal in number. In *infantile scurvy* the hæmorrhagic effusions are in connexion with the shafts of the bones and not with the joints. Hæmophilic joints closely resemble *tuberculous arthritis*, and may also be confused with *rheumatoid arthritis* or *osteo-myelitis*. As death may follow operation, it is important to consider the possibility of hæmophilic hæmarthrosis in cases of obscure joint-disease before active measures are adopted. Aspiration of the joint with a fine needle, which can be performed without risk, will throw light on the diagnosis. Subcutaneous hæmatomas, which are more often seen on the lower extremities, may imitate abscesses.

Treatment. Prophylaxis.—The female members of "bleeder" families should be urged not to marry; however, this counsel is often neglected, and unfortunately they are exceptionally prolific. The patients should be placed under the best hygienic conditions and protected from injury, and should not be operated upon. Residence in a really warm climate appears to exert a favourable influence. The diet should be generous, and the only restriction—and this, perhaps, is somewhat theoretical—is that acids, which by "decalcifying" the blood favour bleeding, should be forbidden. In order to avoid scratches and wounds, all pins and buckles should be banished from the clothes, and boys should not play with pocket-knives. The teeth should be regularly inspected so that early stopping may obviate the need for extraction. Internally, nuclein and ovarian extract have been suggested by Wright to correct the underlying defect in hæmophilia; the nuclein would increase the leucocyte count, while ovarian extract might conceivably confer on male "bleeders" the physiological advantages possessed by the female sex.

To arrest hæmorrhage.—Local applications are naturally employed. The use of fluffy cotton-wool, cobwebs, and matico leaves to promote clotting is usually disappointing, but good results have been obtained from the application of gauze saturated with cephalin. Styptics, such as adrenalin chloride (1 in 1,000) solution, may be painted on, but the vaso-constriction is temporary only. Cold in the form of ice or obtained by means of an ethyl-chloride spray also checks the bleeding for a time. The local application of liquor ferri perchloridi or of the actual cautery is inadvisable. The most powerful and successful

HÆMOPHILIA

method is the physiological styptic advocated by A. E. Wright: an extract of thymus gland is freshly made by pounding it up and mixing it with saline solution (0.5-1 per cent.) to which a little sodium carbonate has been added, the strength being 10 parts of the solution to 1 of the gland. When urgently wanted it may be filtered at once through fine calico; to the filtrate is then added 0.25-0.5 calcium chloride (measured as crystals) and 1 per cent. carbolic acid. When time does not press, the extraction may be continued for 12 to 24 hours, the carbolic acid being added at the start and the calcium chloride as before. If thymus cannot be obtained, testes or the mucous membrane of the stomach may be used, but pancreas should not be employed. The bleeding area should be plugged with wool or lint soaked in the physiological styptic, so as to get it into the closest contact with the bleeding vessel.

Internal remedies may be used to increase the coagulating power of the blood. But the coagulation time should first be determined, for if it is normal no benefit can be expected from this line of treatment. Calcium salts have been extensively employed. According to A. E. Wright, some patients cannot absorb calcium salts, but fortunately they are benefited by magnesium salts. Fifteen grains each of calcium lactate and magnesium lactate should be given at four-hourly intervals for six doses. Calcium salts should not be given hypodermically. The same authority advocates inhalation of CO₂ by the mouth in urgent cases to increase the coagulating power of the blood. Hypodermic injection of horse-serum or human blood (especially from the mother) has been employed with some success. The state of anaphylaxis is associated with increased coagulability of the blood; accordingly, injections of serum from human beings or animals in an anaphylactic condition have been given, and anaphylaxis has been cautiously induced in patients, with encouraging results. Hypodermic injection of gelatin should not be countenanced. Blood transfusion may be employed as a last resort.

For the acute hæmarthrosis absolute rest in bed with an ice-bag should be ordered. After a few days the adjacent muscles, but not the joint, should be massaged; this relieves the pain, promotes absorption, and prevents muscular atrophy. Contracted joints should be treated by extension.

H. D. ROLLESTON.

HÆMOPTYSIS

HÆMOPTYSIS.—Though literally meaning "spitting of blood," the term is usually employed to indicate bleeding from the lungs or respiratory passages.

Etiology. Pulmonary causes.—By far the most important and most common cause of severe hæmoptysis is phthisis. A brisk hæmoptysis may be the first indication of the disease, and the most careful examination may fail to reveal any physical signs. The sputum is frequently blood-tinged, whilst a severe or moderate hæmoptysis may occur at any period during its course and may precede its fatal termination. A large bright-red hæmorrhage may also occur at the onset of pneumonia, but more commonly the "rusty" sputum which contains altered blood is the only indication of bleeding. Sometimes a considerable amount of bright-red blood is intermixed with the sputum throughout a pneumonia. This was especially exemplified in the pneumonia complicating the influenza epidemic of 1917-19. Pulmonary infarction is another cause of hæmoptysis, either considerable or slight. Passive congestion from heart disease, especially mitral disease, is second in frequency only to phthisis as a cause of repeated and serious bleeding from the lungs. Rarer causes of pulmonary hæmorrhage are abscess, hydatid disease, gangrene, and neoplasm. In the last the sputum comes to resemble "prune juice." Slight bleeding may occur in emphysema.

Bleeding from the respiratory passages.—Ulceration of the larynx, trachea, or bronchi may cause hæmoptysis which, though usually slight and recurrent, may be considerable. An aneurysm may "weep" into the trachea or a bronchus, or by rupture lead to sudden death. In bronchiectasis a brisk hæmorrhage occasionally occurs, but slight hæmoptysis is more common. The sputum of acute or chronic bronchitis is sometimes streaked with blood. The endemic hæmoptysis of China and Japan, due to a bronchial fluke, must also be mentioned.

General causes.—Among the rarer causes of hæmoptysis are the hæmorrhagic fevers, purpura hæmorrhagica, scurvy, erythræmia, and severe anæmias and leukæmia. Bleeding from the respiratory system is certainly very unusual in these diseases. Hæmoptysis has been described in elderly people with joint-affectations. Trauma, as from fractured rib, or gunshot or bayonet wounds, is another cause. Of considerable importance is an unexplained severe

HÆMORRHAGE, ANTE-PARTUM

hæmoptysis which occurs in young people without apparent cause. It naturally raises the suspicion of phthisis, but in a large proportion of cases no other symptoms or signs eventuate.

Diagnosis.—The occurrence of hæmoptysis should always lead to a careful examination of the heart and lungs, and inspection of the upper air-passages when the condition of the patient permits it. In the great majority of cases its nature is readily ascertained. The distinction from hæmatemesis is sometimes difficult (*see* HÆMATEMESIS).

Treatment.—Severe hæmorrhage requires immediate treatment. The patient must be kept absolutely at rest in the recumbent position. No attempt to administer food or stimulants is allowable; morphia ($\frac{1}{4}$ to $\frac{1}{2}$ gr. must be injected hypodermically, and a saline purge given by the mouth. When a focus in the lungs is suspected an ice-bag is often applied over it, but is of doubtful value. Vaso-constrictors, such as adrenalin, atropine, and digitalis, increase the amount of blood in the lungs, and are therefore of very problematical value. Ergot is distinctly contraindicated, for it increases the pressure in the pulmonary circulation. Cardiac depressants such as aconite and pituitary extract are more useful. Amyl-nitrite inhalation is sometimes attended by marked success. The induction of artificial pneumothorax has been advocated. After cessation of the bleeding the greatest care must still be exercised in the handling and examination of the patient; the diet should not be stimulating, and alcohol and tobacco are taboo. The blood-pressure should be kept low by saline purgation.

A moderate hæmorrhage from heart disease needs no treatment and may be beneficial.

For protracted hæmorrhages of small amount the measures indicated are those to lower blood-pressure and increase the coagulability of the blood. For the latter purpose calcium chloride or calcium lactate (20 gr.) every four hours, or blood-serum or normal horse-serum, may be employed. Treatment must, of course, be directed to the cause.

FREDERICK LANGMEAD.

HÆMORRHAGE, ANTE-PARTUM.

The literal meaning of ante-partum hæmorrhage is, of course, uterine hæmorrhage during pregnancy, whatever the period of pregnancy and whatever the source of the bleeding. But, as used in obstetrics, the term is limited to denote bleeding from the placental site during the last twelve

weeks of pregnancy or during labour before the birth of the child. During the first twenty-eight weeks of pregnancy, hæmorrhage from separation of the placenta or ovum is classified under abortion. The division is not a scientific one but is made merely for the convenience of clinical classification. For example, implantation of the placenta in the lower uterine segment (placenta prævia) often causes hæmorrhage before the twenty-eighth week of pregnancy—in fact, placenta prævia is a not uncommon cause of abortion; thus, a case of placenta prævia with early hæmorrhage would be called a case of "abortion," whereas if the hæmorrhage were postponed until after the twenty-eighth week of pregnancy the case would be called one of "unavoidable ante-partum hæmorrhage," although in both the cause would be the same.

Minor causes of uterine hæmorrhage during pregnancy are carcinoma of the cervix and mucous polypi; bleeding from these causes is slight, and the diagnosis is obvious when the patient is examined.

Varieties.—There are two varieties of ante-partum hæmorrhage. (1) Hæmorrhage due to the detachment of a normally situated placenta is known as *accidental hæmorrhage*; (2) hæmorrhage due to the detachment of a placenta prævia is known as *unavoidable hæmorrhage*.

1. ACCIDENTAL HÆMORRHAGE

Etiology and pathology.—If very slight cases be included, accidental hæmorrhage is found to occur about once in 500 pregnancies. It is very rarely due to an accident (blows or falls) as its name might imply. In fact, "accidental," in the usual sense of the word, is a misnomer when applied to this variety of hæmorrhage. The two varieties of ante-partum hæmorrhage were named "accidental" and "unavoidable" by the British obstetrician, Rigby; he used the word "accidental" as implying "fortuitous" or "without cause," in contradistinction to the obviously "unavoidable" nature of the hæmorrhage in the presence of placenta prævia.

A very small percentage of cases of accidental hæmorrhage is due to mechanical causes such as gross trauma or the dragging of a very short umbilical cord. But modern observations show that about 80 per cent. of the patients have albuminuria of greater or less degree, whilst a few have œdema and other generalized symptoms of the toxæmia of pregnancy. The clinical evidence that accidental hæmorrhage

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has a toxæmic origin is overwhelming; and the clinical is further strengthened by the pathological evidence that in many fatal cases necrotic and hæmorrhagic changes in the liver and kidneys have been found, similar to the well-known changes in eclampsia. The placental decidua in cases of accidental hæmorrhage is invariably found in a state of patchy necrosis, and herein lies the primary source of the hæmorrhage. The decidual vessels, which are mere channels in the decidua, give way under the combined effect of necrotic changes in their walls and supporting tissues and the high blood-pressure which almost invariably accompanies these cases. This leads to retroplacental hæmorrhage, which may be the starting-point of detachment of the placenta from the uterine wall over a wide area, and profuse hæmorrhage; the blood either accumulates within the uterus, between the uterine wall and the detached placenta or membranes ("concealed" hæmorrhage), or, as is more usual, tracks down behind the membranes and escapes through the cervix ("external" hæmorrhage). In other cases, slighter and more fortunate, the initial retroplacental hæmorrhage and consequent placental separation is much less extensive; nothing more than a "retroplacental hæmatoma" is formed. The mother seldom has symptoms of such an event, but the fœtus usually dies from "relative placental insufficiency;" subsequent examination of the placenta in such cases reveals a cup-shaped depression on the maternal surface occupied by old, sometimes laminated, bloodclot.

In concealed hæmorrhage (*see below*) the uterus is subjected to a considerable bursting-stress by the accumulation of blood within it. The overstretched muscle and other tissues give way, and widespread interstitial hæmorrhages may be found throughout the walls of the uterus; the blood, tracking towards the peritoneal coat, often forms subperitoneal hæmorrhages, and may infiltrate between the layers of the broad ligament, constituting a considerable hæmatoma. Sometimes the peritoneal coat splits under the stress, and occasionally the peritoneal cavity is found to contain a fair amount of blood.

Clinical varieties.—Accidental hæmorrhage may be (1) external, (2) internal or concealed, (3) mixed external and concealed.

When the placenta becomes separated, the rise of intra-uterine pressure due to the retroplacental hæmorrhage usually stimulates the uterus to a state of activity; the uterine

contractions drive out the blood, which tracks down behind the membranes and escapes through the cervix. This is *external accidental hæmorrhage*. The uterine contractions have another most important effect: they control both the hæmorrhage and the tendency to further separation of the placenta.

If, on the other hand, the uterus remains inactive or is paralysed by the sudden distension due to a very copious initial retroplacental hæmorrhage (as would occur if the initial placental separation were over a wide area), the blood is retained in the uterus. This is *concealed accidental hæmorrhage*, and is perhaps one of the most dangerous complications of midwifery: in such cases the placental separation is progressive, and very often the whole placenta becomes detached.

In many cases the hæmorrhage is partly external and partly concealed—the *mixed* variety.

A special variety, already referred to, is the formation of a small *retroplacental hæmatoma*; the separation of the placenta is sufficient to kill the fœtus, but the amount of hæmorrhage is not enough to produce maternal symptoms. In these cases the fœtus is born in a state of maceration, and the true state of affairs is only revealed by the discovery on the maternal surface of the placenta of a deep depression occupied by an old bloodclot.

Symptomatology. *External accidental hæmorrhage.*—There is usually no obvious cause for the bleeding; the amount lost varies from a mere "show" to a very copious hæmorrhage. The patient is not in pain. On abdominal examination, there are no abnormal physical signs. On vaginal examination, there may be clots in the vagina, and, if the finger can be passed through the cervix, bloodclot is usually felt in the lower uterine segment; the placenta cannot be felt. The urine (catheter specimen) may contain albumin—a trace or more. There may be other signs of pregnancy toxæmia, such as œdema and raised blood-pressure. If the fœtal heart-sounds cannot be heard, the inference to be drawn is that there has been sufficient placental separation to kill the fœtus.

Concealed accidental hæmorrhage.—There are few clinical states more characteristic than a severe case of concealed accidental hæmorrhage; it is difficult to mistake it for anything else. The patient has the signs and symptoms of severe internal hæmorrhage and shock in combination with those due to sudden over-

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distension of the uterus. There are pallor, sweating, restlessness, sighing respirations perhaps, a rapid small pulse, and severe and continuous abdominal pain from the uterine distension. On abdominal examination, the uterus is larger than it should be, feels uniformly hard and solid, is very tender all over, and the fœtus cannot be distinguished; as the fœtus is always dead, the fœtal heart cannot be heard. As a more or less severe state of pregnancy toxæmia almost invariably accompanies these cases, the general condition of the patient is correspondingly worse; the urine is usually scanty, and contains a large amount of albumin and tube-casts. On vaginal examination, the cervix, as a rule, is found closed.

In cases in which the internal hæmorrhage is less profuse the patient will present the above state on a diminished scale. But it is characteristic of these patients that they are much more ill than is to be accounted for by the amount of hæmorrhage; this is because the over-distension of the uterus causes severe shock. Cases of moderate severity are sometimes seen in which the pulse-rate remains slow, though the volume is small and the patient is pallid; this is puzzling and deceptive, and may lead the unwary astray.

In the worst cases of concealed hæmorrhage there is no external hæmorrhage whatever: this is implicit in what has already been written. Inactivity or paralysis of the uterus leads not only to retention of the blood but also to progressive placental separation and hæmorrhage. Activity of the uterus is proclaimed by the appearance of external hæmorrhage, which is therefore a very favourable sign.

Mixed concealed and external accidental hæmorrhage.—These cases share the characters of both the other varieties; they may be said to include the less severe cases of the concealed and the more severe cases of the external variety. In the former, as soon as the uterus recovers from its initial shock, external hæmorrhage invariably appears; in the latter, although the retained blood may not be in sufficient quantity to make the uterus characteristically enlarged and hard, it reveals itself after the birth of the fœtus by a mass of dark clot accompanying or following the expulsion of the placenta.

Diagnosis.—External accidental hæmorrhage has to be distinguished from *unavoidable hæmorrhage* (placenta prævia). This can only be done with certainty by failing to feel the placenta on vaginal examination. Cases of

low implantation of the placenta ("lateral placenta prævia") are often mistaken for cases of external accidental hæmorrhage. After delivery, the distinction can always be made by examining the placenta and membranes; if the case has been one of placenta prævia, the opening in the membranes through which the fœtus has passed should be close to the edge of the placenta. Two secondary diagnostic points often help: in accidental hæmorrhage there is usually albuminuria and the fœtus is usually dead.

Prognosis.—*Concealed* accidental hæmorrhage is probably the most dangerous accident in midwifery. About 20 per cent. of the mothers and all the fœtuses die, the former from hæmorrhage and shock, the latter from asphyxia due to separation of the placenta. In these cases the appearance of external hæmorrhage is of good prognostic import, as it indicates the onset of uterine contractions and therefore shows that the case is taking a favourable turn. In cases of *mixed* hæmorrhage, and still more in cases with *external* hæmorrhage only, the prognosis is much more favourable. Still, accidental hæmorrhage of any sort should lead to a guarded prognosis. Everything depends, of course, on the type of case. In a multipara with a dilating cervix the outlook is much more favourable than in a primigravida not in labour; the more premature the fœtus, and the easier its delivery, the better the outlook. An important point to bear in mind is that the seriousness of a case must not be judged merely by the amount of blood lost externally; the general condition of the patient and the physical state of the uterus are far more important, indicating as they do the presence or absence of a serious internal hæmorrhage.

Treatment.—In all cases of accidental hæmorrhage, of whatever variety, the great principle of treatment is to excite vigorous labour pains; the blood is coming from that part of the uterus (upper uterine segment) which is capable of contraction and retraction, and the bleeding is nearly always controlled when strong labour is established. No one method of treatment is applicable; in general terms, the form of treatment will depend on the slowness or severity of the bleeding, on whether the bleeding is external or concealed, on the presence or absence of labour pains, on the degree of dilatation of the cervix, and on the general condition of the patient. Cases of external hæmorrhage may be considered, for

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purposes of treatment, under the headings of *slight*, *moderate* and *severe*; concealed hæmorrhage, which is always severe, stands in a class by itself.

External hæmorrhage.—Many cases of external hæmorrhage are extremely *slight* and require no active treatment. Keep the patient in bed and give mild sedative drugs; the bleeding will probably stop and the patient deliver herself in perfect safety; or, if premature, the pregnancy may go on to full term without any recurrence of bleeding. In such cases, of course, a careful lookout must be kept on the patient; her general condition, her pulse-rate, and the state of her uterus must be watched, to exclude concealed hæmorrhage. Always test the urine, and give appropriate treatment if there is albuminuria.

Cases of *moderate* severity are those in which the bleeding is too severe to justify temporizing measures but not severe enough to endanger life. The treatment depends essentially upon whether the patient is in labour or not; the object of treatment is to excite vigorous labour pains. If the patient is in labour and the cervix is dilating, rupture the membranes, apply a firm abdominal binder, and give an intramuscular injection of pituitary extract (1 c.c.); the injection may be repeated four-hourly. Once strong and regular labour pains have started, there is little or no fear of further hæmorrhage. But if hæmorrhage continues, the dilatation of the cervix must be completed with the hand or hastened by the insertion of a large Champetier de Ribes bag, and delivery completed with the forceps. Violent and hasty methods of delivery must be avoided, for, once strong labour is established, there is seldom any cause for anxiety, and the condition of the patient may be such that she cannot withstand any additional shock or injury.

If the uterus is not active, and there are no labour pains, the case is much more dangerous. Rupture of the membranes must be avoided, for it causes an immediate lowering of the intra-uterine pressure and encourages further hæmorrhage. Forced delivery by manual or instrumental dilatations of the cervix or by version will probably be fatal to a patient already in a state of collapse. The best treatment for these cases is to plug the vagina, apply a firm abdominal binder, and inject pituitary extract. Vaginal plugging, if properly carried out, will arrest the bleeding in about 90 per cent. of cases, but it is not infallible. Half-hearted plugging is useless. An

anæsthetic, an assistant, the lithotomy position, and rigid asepsis are essential. Better dispositions cannot be given than those of Hastings Tweedy. "To plug efficiently, the left hand should be passed into the vagina with the palmar surface directed towards the hollow of the sacrum, while the tips of the fingers lie behind the cervix. Small pieces of cotton-wool, squeezed out of lysol solution, and each the size of the thumb-knuckle, are then taken and inserted by means of the right hand round the cervix. The fingers of the left hand are kept busy squeezing the pellets into a compact mass and forcing the spaces between them to permit the insertion of still another plug. This process is continued in a systematic manner from above downwards till the vulva is reached and the vagina can hold no more. A T-bandage is applied to keep the plug in position, and an abdominal binder is fastened tightly from above downwards to press the side walls of the uterus against the vaginal dam, and this completes the operation. A plug so applied will cause immediate cessation of hæmorrhage, and, when it is removed after the lapse of hours, so much blood only will be found as can be accounted for by the flow which took place during the operation.

"The vaginal plug is not easy to apply, nor is its application harmless. Sometimes, in spite of our best efforts, we cannot at the first attempt insert sufficient material to stop the bleeding. In such cases the plug must be removed entirely and reinserted. On the second attempt the procedure will be found to have become much more easy on account of the dilatation of the vagina."

Pain and a certain amount of shock always follow the application of the plug, and these must be relieved by morphia. The plug acts partly by exciting labour pains, but principally by exerting pressure on the uterine arteries. It should be removed a few hours after labour pains have begun, as, if left in too long, it will obstruct delivery.

In cases in which the hæmorrhage is *severe*, there are usually no labour pains and no dilatation of the cervix, for hæmorrhage is not likely to be severe if the uterus is active. The treatment for severe cases is the same as that already given for moderate, with the additional warning that hasty delivery will be surely fatal to an already collapsed patient. A vaginal plug and injections of pituitary extract, repeated four-hourly, will control the bleeding and tide the patient over the next few hours.

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during which time she must be treated for collapse and shock.

Where the necessary skill and surroundings are available, Cæsarean section is the best treatment for many cases of accidental hæmorrhage, especially when the patient is a primigravida and the cervix is closed. Spinal anæsthesia should be used in the presence of collapse, both because it tends to prevent shock and because of its remarkable action in exciting uterine contraction.

Concealed hæmorrhage.—In concealed hæmorrhage the uterus, as we have seen, is always inactive. The invariable presence of albuminuria and other toxicæmic symptoms adds to the danger of the condition.

The ideal treatment for *severe* concealed hæmorrhage is Cæsarean section, preferably under spinal anæsthesia. It is not necessary to remove the uterus, as it will always contract if given time and stimulation; nor do interstitial hæmorrhages impair its value as a future child-bearing organ. If Cæsarean section is not available, these cases must be treated by the vaginal plug and pituitary extract. Active treatment, by Cæsarean section or the vaginal plug, is not always necessary. In cases of *moderate severity* nothing need be done beyond giving pituitary extract, the patient's general condition and pulse-rate and the size and state of her uterus being carefully watched. Very often the general condition improves and labour pains start, a fact that is proclaimed by the appearance of external hæmorrhage, and the patient delivers herself safely. It requires great judgment to adopt this line of treatment, and should only be done in the presence of all facilities for instant operative treatment should there be signs that hæmorrhage is progressive.

2. UNAVOIDABLE HÆMORRHAGE (PLACENTA PRÆVIA)

When the placenta is implanted, wholly or in part, in the lower uterine segment, it is called a *placenta prævia*. The lower uterine segment has no anatomical upper boundary; it is merely that part of the uterus which has to dilate and stretch during labour to allow the exit of the fœtus; it comprises the lower three inches, roughly, of the uterine cavity, as measured upwards from the internal os. As soon as labour begins, the uterine contractions cause the cervix and lower uterine segment to dilate and to be drawn or retracted upwards, and at the same time cause the presenting or lower pole of the ovum—the bag of waters—

to advance or slide downwards; in *placenta prævia* the placenta is involved in this downward movement of the lower pole of the ovum, and is dragged or "sheared" off the retracting lower uterine segment. Hæmorrhage from the placental site is in such circumstances inevitable; hence the aptness of the name "unavoidable" given to this variety of hæmorrhage.

Etiology and pathology.—*Placenta prævia* takes place about once in five hundred labours. It is naturally more frequent in multigravidæ than in primigravidæ, because the former is a much more numerous class than the latter. Why, in certain cases, the ovum should get implanted in the lower instead of in the upper uterine segment is not clear. Such cases are perhaps best regarded as instances of "delayed implantation" of the ovum. In normal circumstances the ovum enters the uterine cavity from the Fallopian tube, where it has been fertilized, when it has reached a certain stage of development—when it has acquired an active trophoblast and is capable of burrowing into the uterine decidua as soon as, or very shortly after, it meets it. But if for any reason it happens to arrive in the uterine cavity in a less advanced stage of development, its trophoblastic activity may not bring about its embedding until it has travelled downwards as low as the lower uterine segment or cervix.

Separation of a *placenta prævia* and hæmorrhage often occur before labour has started; the reason for this is that, during the last weeks of pregnancy, painless uterine contractions often bring about an expansion of the lower uterine segment and a partial opening up of the cervix. It is common enough in normal cases, towards the end of pregnancy, to find the cervix completely taken up and even the external os slightly open. Such an occurrence is bound to be accompanied by slight advance of the lower pole of the ovum and, if the placenta happens to be *prævia*, a certain amount of separation from the uterine wall is bound to occur. A *placenta prævia* usually occupies a wider area, is thinner than normal, and is often irregular in shape; this is because the decidua is thin in the lower uterine segment and the placenta cannot obtain such a good blood supply. There is often a marginal or velamentous insertion of the umbilical cord; large areas of degeneration and "white infarction" are common, and there may be areas of morbid adhesion to the uterine wall.

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The source of the hæmorrhage in placenta prævia is the maternal blood sinuses of the placental site; the placenta itself does not bleed from its bared maternal surface, or bleeds only slightly and momentarily. But if large umbilical branches are torn through, the fœtus may bleed to death; such an event may occur under two conditions: (1) In cases of velamentous insertion of the umbilical cord, "vasa prævia" may course along the area of amnion forming the bag of waters and may be torn when the amnion ruptures; (2) in cases of complete placenta prævia, when the placenta is perforated or torn through by the accoucheur, large umbilical vessels on the fœtal surface of the placenta may be ruptured.

There are two additional points of great practical importance in placenta prævia: (1) the lower uterine segment is thicker and more vascular, does not dilate well, and is more easily torn by manipulations; (2) the placental site lies very close to the vulvo-vaginal tract, and is especially accessible to bacteria from this source.

Varieties.—The clinical varieties of placenta prævia are named according to the relationship of the placenta to the internal os uteri, as felt on vaginal examination. There are two main varieties, complete and incomplete. In **complete** placenta prævia the placenta is felt completely covering the internal os. In the **incomplete** variety the os is not completely covered by the placenta. Cases of incomplete placenta prævia may be divided into sub-varieties such as (a) *partial* placenta prævia, when the os is partly covered by placenta; (b) *marginal*, when the lower edge of the placenta just reaches the margin of the os; (c) *lateral*, when the lower edge of the placenta does not reach as low down as the os but is felt by the examining finger a short distance above it. In the two latter sub-varieties the placenta lies only partially in the lower uterine segment. The most complete variety of all is when the middle of the placenta lies over the internal os: this is known as *central* placenta prævia. It is obvious that this degree of completeness cannot be determined when the os is only slightly dilated; often it is only apparent after labour, when an opening will be found in the placenta through which the fœtus has passed; or during operative manipulations, when the accoucheur will fail to reach the edge of the placenta in any direction.

An inspection of the placenta and membranes after delivery will reveal the position

the placenta has occupied. The closer the opening in the membranes to the placenta, the lower has been its position in the lower uterine segment; in cases of complete placenta prævia the placenta itself will be found torn through; and in cases of central placenta prævia the opening may be found occupying the placenta itself.

Symptoms.—The only symptom is hæmorrhage. "A painless, causeless hæmorrhage in the last three months of pregnancy is almost pathognomonic of placenta prævia." The diagnosis is, of course, verified by making a vaginal examination and feeling placental tissue over or near the internal os. The first hæmorrhage is usually a slight one; but cases have been known where it was so violent as to be fatal. After an interval of hours or days the hæmorrhage is repeated; repeated hæmorrhages are characteristic, and each succeeding one is apt to be greater. Unless the case be treated at this stage a profuse, even fatal, hæmorrhage is likely sooner or later to occur; the effect of repeated small hæmorrhages is to produce a very profound state of anæmia, and to place the patient in an unfavourable state for withstanding the collapse of a profuse hæmorrhage or the shock of operative treatment, or for resisting a septic infection in the puerperium.

As a rule, the more complete the placenta prævia, the earlier the hæmorrhage begins; in cases of marginal implantation the hæmorrhage is often postponed until labour has started, and is usually less profuse than in the complete variety. Central placenta prævia is the most dangerous of all, the bleeding appearing early and nearly always being profuse and continuous.

The occupation of the lower uterine segment by the placenta interferes with the accommodation of the fœtal head; in consequence, breech and transverse presentations are common. As will be pointed out later, the presentation of the breech is a positive advantage from the point of view of treatment.

Diagnosis.—A hæmorrhage, especially if repeated, near the end of pregnancy is always strongly suspicious of placenta prævia. There must be no delay in confirming the diagnosis by feeling the placenta through the cervix; as the cervix is usually open enough to admit the finger, this is easy enough in most cases; but if the cervix is closed, so important is it to make a correct diagnosis that the patient should be anæsthetized and the cervix dilated enough to

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admit the finger. The possibility that labour may thereby be induced is no objection and is a risk that must be taken; for if the case is one of placenta prævia, as it most probably is, labour will have to be induced in any case. There is no other way of making a sure diagnosis. It is true there are other suspicious signs, such as feeling through the vaginal fornix that something thick intervenes between the presenting part and the examining finger; while some people claim to be able to divine the presence of the placenta in the lower uterine segment by abdominal palpation. The only condition which enters seriously into the diagnostic competition is *accidental hæmorrhage*; the chief point here, of course, is that the placenta cannot be felt, even if the finger be introduced far through the cervix. But there are two other helpful points: in *accidental hæmorrhage* there is usually albuminuria, and the placental separation has usually been wide enough to kill the fœtus. Too much reliance must not, however, be placed on albuminuria as a diagnostic point, as it not infrequently accompanies cases of placenta prævia.

Prognosis.—When a patient has placenta prævia her friends had better be told at once that she is in a position of considerable danger and that the fœtus has but a small chance of survival. The general prognosis as regards life is well given by Ellice MacDonald's mass-figures: he collected 10,600 cases and found a maternal mortality of 7·7 per cent. and a fœtal mortality of 55 per cent. This collection of cases extended over many years (in 1887, for example, the maternal mortality was 23 per cent.) and included all types of cases treated with varying degrees of skill. But in few other conditions does the outcome of an individual case depend more upon its individual circumstances. The following are the factors which chiefly affect the prognosis, viz. the variety of placenta prævia, the skilfulness of the treatment and the methods employed, and the condition of the patient when first seen, i.e. the amount of blood she has already lost, if she is in labour or not, and the degree of dilatation of the cervix. The causes of *maternal death* are hæmorrhage from the placental site, injury and shock during operative delivery, post-partum hæmorrhage, and puerperal sepsis. The causes of *fœtal death* are asphyxia from separation of the placenta or from profound maternal anæmia, fœtal hæmorrhage from torn placental umbilical vessels, prolapse of the cord, and intracranial injury and hæmorrhage

consequent on rapid delivery after version. Prematurity is also an important factor in the fœtal mortality. As regards variety, the complete is very much more dangerous than the incomplete and marginal, both for the mother and for the fœtus; the fœtus, in fact, seldom survives in a complete case. The reasons for this are that the area of placental separation is necessarily wide, that the membranes are out of reach, that the dilatation of the cervix is interfered with, and that operative manipulations are bound to disturb the placenta still more and to cause more hæmorrhage. Complete placenta prævia in a primigravida with a closed and rigid cervix is one of the worst complications of pregnancy. On the other hand, in the incomplete, and especially in the marginal varieties, the membranes can be reached, the dilatation of the cervix is not much interfered with, and operative manipulations need not further disturb the placenta. Some of the most hopeless cases are those in which the patient is profoundly anæmic before treatment is begun; many patients are lost by neglecting the rule that, at the first hæmorrhage, the diagnosis must be made and treatment begun at once. Many of these neglected cases die from the shock or additional hæmorrhage of operative delivery. If the patient is already in labour and, better still, if the os is well dilated and supple, the ease of treatment is enormously increased.

The prognosis as regards the particular method of treatment adopted is not easy to appraise, because of the wide variation in the nature of cases of placenta prævia. Cæsarean section is becoming increasingly popular; as regards the mother, the results obtained are no better than, if so good as, those got by skilful midwifery, except in a narrow category of cases; as regards the fœtus, the results are, of course, extremely fine. There does not seem to be much difference, as regards the mother, between version and the hydrostatic bag; if anything, the maternal mortality is rather lower after version. As regards the fœtal mortality, with a bag it is about 40 per cent., whereas with version it is about 60 per cent.

Treatment.—One thing the practitioner should, with one exception, never fail to do; he should begin active treatment directly he has made the diagnosis. The temporary cessation of hæmorrhage may give a false sense of security and an excuse for temporizing. Let him never fall into this trap; a profuse hæmorrhage may come at any moment. The one

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circumstance which makes it best for a while to postpone active treatment is when the patient is very collapsed from hæmorrhage and the hæmorrhage has temporarily ceased. In such a situation immediate active treatment might kill the patient from shock, and the period of freedom from hæmorrhage should be used for restoring her by warmth and saline infusions; all the time she must be continuously watched for a return of hæmorrhage, and everything likely to be required for active treatment must be in readiness.

Let the practitioner also bear in mind the causes of maternal death, as enumerated under Prognosis—hæmorrhage from the placental site, the shock or injury from hurried delivery, post-partum hæmorrhage, puerperal sepsis.

The great principle in the treatment of placenta prævia is to maintain continual pressure on the placental site until the patient can be safely delivered. (The cases suitable for Cæsarean section will be considered later.) Pressure on the placental site is secured in two ways: (1) by bringing down a leg so as to plug the lower uterine segment with the half-breech, (2) by introducing a Champetier de Ribes bag through the cervix. Before either of these methods can be used, the cervix must have reached a certain degree of dilatation—in fact it must be large enough to admit two fingers with ease; the practitioner can then employ one of the two pressure methods. But when the cervix is closed he is faced with the preliminary problem of dilating it. Here he has two methods to rely upon: either he may dilate with metal dilators up to size No. 26 on Hegar's scale, or he may employ the slower method of plugging the cervix with sterile gauze and applying a firm abdominal binder. For either of these little surgical operations an anæsthetic and an assistant are necessary and strict aseptic precautions must be taken. The plug controls hæmorrhage by direct pressure and excites pains; it must not be left in for more than eight hours, by which time the cervix will be found sufficiently dilated.

The alternative methods of bag and version may now be compared. On the whole, version is undoubtedly the method of choice; it is suitable for all varieties of placenta prævia, it is a more reliable means of effecting pressure, it gives absolute control over labour, it requires no apparatus, and most practitioners are more accustomed to it than to the bag. The patient is liable to bleed when the bag comes out unless the presenting part descends at once of its own

accord or is brought down artificially; after the introduction of a bag, therefore, she requires constant supervision. Herein the bag compares badly with version; for after version no other operation is necessary to effect delivery: the fœtus is delivered as a breech, either spontaneously or by traction on a leg, and continuous pressure is maintained on the placental site from the moment of bringing down the leg until complete delivery. Experience is most emphatically against the use of the bag in cases of complete, and especially of central, placenta prævia; the reasons for this are that hæmorrhage will always have been severe in such cases and no risk whatever must be run of recurrent bleeding, and that version is the easiest method of transplacental delivery. Lastly, bags tend to displace the presenting part, deteriorate by keeping, and sometimes burst at critical moments.

Were it not that the foetal mortality is about 20 per cent. lower with their use, bags would never enter into competition with bringing down a leg in the treatment of placenta prævia. But the life of the mother is the first consideration, and version is more favourable to her. If the practitioner is expert in the use of the bag, let him by all means use it in *marginal* and *lateral* cases where the membranes are in reach and the bag can be pushed through them. In these cases the maternal risks are no greater with the bag than with version. The bag must, of course, lie in the amniotic cavity, and the membranes, if intact, must be ruptured before its introduction.

On the method of pulling down a leg.—When the fœtus is presenting by the breech, this is simple enough. Two fingers are introduced through the cervix, the membranes are ruptured or the placenta is perforated, as the case may be, a foot is taken hold of and the leg drawn down. When unfortunately the legs are found extended, the whole hand has to be passed up to the fundus of the uterus.

In vertex presentations the fœtus must be turned, and there are three ways of doing this: (1) external version, (2) bipolar version, (3) internal version.

External version is incomparably the best of these, and is usually easy, since the head is very seldom engaged; it is most suitable for cases seen early, with intact membranes. After the breech has been got into the lower uterine segment, two fingers are introduced through the cervix and a foot is drawn down. Failing external version, the bipolar method of

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Braxton Hicks must be performed. Failing both these, nothing is left to do but to bring down a leg by *internal version*. This has serious disadvantages, and must always be regarded as a last resort. The cervix must be sufficiently dilated to admit the hand. The disadvantages to the mother are that the placenta may be further detached, that there is a risk of introducing infection, and that the operation causes a certain amount of shock. The serious risk to the foetus is that prolapse of the cord is very likely to accompany the operation.

When the placenta completely covers the os uteri, an attempt must always first be made to reach the membranes at one side; this failing, the placenta itself must be perforated in order to bring down a leg or to introduce a bag. This should be done in such a way as to produce a minimum of further placental detachment; the hand must not be pushed forcefully through the placenta, but it must be gently and gradually bored through with two fingers. In all operative manipulations the patient should be in the dorsal position with the legs held by an assistant or by a (Clover's) crutch, and the operator seated in front of her.

After the leg has been brought down, or a bag has been introduced, it should gently be pulled on, so as to stop bleeding by thoroughly plugging the lower uterine segment and compressing the placental site. This may be done by an assistant; or continuous weight traction may be exerted by attaching a weight of 2 lb. to a tape fastened to the leg or bag. Only gentle traction must be exerted, not more than enough to control the bleeding.

The warning against hurried delivery in placenta prævia cannot be emphasized too much. *Slow delivery should be the invariable rule*. Hurried delivery is not only dangerous for the foetus but, worse still, is very likely seriously to injure the mother by causing extensive lacerations of the cervix and lower uterine segment; such lacerations, involving as they usually do the placental site, not only provoke severe hæmorrhage, but greatly increase the risk of infection. Again, when a patient is suffering from the effects of hæmorrhage, the shock and additional hæmorrhage of hurried delivery may turn the scale against her. Once hæmorrhage has been controlled, there is no need for hurry; the slower the patient is delivered the better.

Treatment of slight cases.—Many cases of placenta prævia are slight, and it is by no

means always necessary to treat them by bringing down a leg or by inserting a bag. By a slight case is meant one in which the placenta is marginal or lateral, the bleeding is slight, the patient is having good pains, and the os is dilating well. For many such cases no more need be done than to rupture the membranes. This enables the placenta to follow the retracting lower uterine segment without much further detachment, and allows the presenting part to descend and compress the placental site. In such cases labour, as a rule, progresses rapidly and the patient delivers herself spontaneously.

Dangers of post-partum hæmorrhage.—After the patient has been safely delivered, all danger is not yet over; for post-partum hæmorrhage frequently occurs from imperfect retraction of the lower uterine segment. A careful look-out should be kept for this, and on the first sign of hæmorrhage the placenta should be expressed or, failing this, removed manually. Hæmorrhage may continue after the placenta has come away, and the usual remedies should be adopted, i.e. intramuscular injections of pituitrin or ergot, hot intra-uterine douche, or bimanual compression. If these fail, the whole uterus should be packed without delay; for it must be remembered that even a slight post-partum loss may prove too much for a patient who is anæmic from ante-partum hæmorrhage. A copious antiseptic intra-uterine douche should be given after labour in all cases of placenta prævia in which there have been intra-uterine manipulations.

Cæsarean section for placenta prævia.—Cæsarean section is becoming increasingly prominent in the treatment of placenta prævia. It is especially indicated in cases of complete placenta prævia in primigravida where labour has not started and the cervix is intact and resistant. Such cases are of a very dangerous type for the mother, and are invariably fatal to the foetus, if treated by the ordinary methods. But, given a capable operator and the proper environment, Cæsarean section need not be limited to that type of case. Provided the patient is in good condition, the mortality of Cæsarean section for placenta prævia should be no higher than that for contracted pelvis. The mortality of the latter is about 3 per cent. for clean cases; and the mortality for cases of complete placenta prævia, treated by conservative methods, even in the best hands, is certainly higher than this. Generally speaking, Cæsarean section is indicated for the

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following type of case: the patient at or near term, in good condition, not in labour and the cervix closed, the placenta completely covering the internal os, the foetus alive. Too much importance should not be given to the foetus, since the operation is undertaken primarily in the interests of the mother; but in cases where she is particularly anxious to have a live child, as for example in a primigravida of over forty, the foetal indication becomes a strong one.

EARDLEY HOLLAND.

HÆMORRHAGE, INTRACRANIAL (see HEAD INJURIES).

HÆMORRHAGE, POST-PARTUM.—

Post-partum hæmorrhage is the inclusive term given to all hæmorrhages issuing from the vagina, from the time of the delivery of the child to the end of the puerperium. Such hæmorrhages may therefore arise from the uterus, before or after the birth of the placenta, or from laceration in the parturient canal.

Hæmorrhages are termed "late" when they occur after the first twenty-four hours.

Etiology.—In the normal uterus, hæmorrhage from the open uterine sinuses is stopped by the retraction and contraction of the uterine walls, aided by clotting of blood in the sinuses. Any condition interfering with retraction and contraction allows hæmorrhage to occur. Hence, partial separation of the placenta, overstretching of the uterus, as in hydramnios, general lack of tone, either primary or through exhaustion, the presence of fibroid tumours, etc., are all potential in producing post-partum hæmorrhage. Lack of clotting in the sinuses occurs in certain general and blood diseases, but is a less important factor.

Laceration may occur in the cervix uteri, in the vagina, or at the vaginal outlet. Deep tears in the cervix, which are particularly liable to occur where there has been low implantation of the placenta (placenta prævia) because the tissues are then more congested and friable, laceration of varices and tears of the clitoris or bulb, may give rise to very severe, even fatal hæmorrhage. Other tears do not often bleed seriously.

Symptoms.—The symptoms of post-partum hæmorrhage are local and general. Locally, blood gushes from the vagina. If the uterus has been controlled through, and for some minutes after, the third stage of labour, as advised under Prophylactic Treatment, the blood is always visible. Sometimes, however,

sudden dilatation of the uterus occurs some time after delivery, in which case attention may first be called to the fact that there is hæmorrhage within the tract by the pallor of the patient and the quickening of her pulse. This quickening of the pulse is not constant. It may remain comparatively slow and then suddenly become more rapid when considerable loss has occurred. Further general symptoms produced by excessive loss of blood are clamminess and chill of the extremities, restlessness, and dyspnoea—"air-hunger." The patient complains of faintness, dizziness, and thirst. Collapse occurs later.

Diagnosis.—Post-partum hæmorrhage will speak for itself, except in those few cases in which the blood is retained in the parturient canal, in which case, also, as soon as the abdomen is examined and the uterus manipulated, blood gushes from the vagina. The determination of the origin of the hæmorrhage, however, is not always easy. With the placenta *in situ*, by keeping one hand on the uterus, observation may be made of the more external parts. When the placenta has come away, if the uterus is firm and small, the diagnosis of laceration must be made and its situation found. The bleeding has sometimes a double origin.

Prognosis.—Hæmorrhage amounting to over one pint is considered pathological. Some patients can bear enormous losses without succumbing; many can bear the loss of 2½–3 pints. Much depends on the patient's general condition and whether there has been loss before or during the earlier stages of labour. Excessive hæmorrhage occurs in about 2 per cent. of deliveries, but in institutions, where effective treatment can be undertaken at once, death from post-partum hæmorrhage is rare.

Thrombosis, embolism, or sepsis complicates the puerperium in many cases.

Treatment.—Stress must be laid on the overwhelming importance of asepsis. Patients who have lost excessively are already prone to sepsis, and the internal manipulations involved are among the most dangerous for infecting the uterus. Thorough disinfection of the hands should be practised, using biniodide of mercury 1 in 1,500 as an antiseptic, and rubber gloves should be worn. The external genitals and vagina should also be well disinfected (externally, biniodide of mercury 1 in 1,500, or tincture of iodine; internally, lysol solution 1 per cent.) before internal exploration or

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treatment is begun. These precautions must be taken in spite of severe hæmorrhage.

Prophylactic treatment.—Attention should be paid to the patient's general condition during the ante-natal period, and treatment given accordingly.

During labour, delivery should not be attempted until the cervix is fully taken up and dilated. In all cases in which forceps must be used or other mechanical aid given, the bladder should first be emptied with the catheter. A thorough examination of the presenting part should then be made, even if this involves introducing the hand into the vagina and feeling around the presenting part. The direction of the ear, for instance, gives great help when a posterior lie is suspected. Adjustment of the presenting part, before the application of forceps, makes the greatest difference in the ease of delivery, and hence reduces the risk of laceration to a minimum. For the same reason, before traction is applied, a final examination should be made to see that nothing but the presenting part has been included in the forceps' grip. Time should be taken in delivery, the pulling, when possible, being with the pains. The uterus should be followed down.

As soon as the child is delivered and separated, the mother should be placed on her back, the knees flexed and thighs separated, with a receiver containing the cord and its clip between them. Control should be taken of the uterus by enveloping it with the hand through the abdominal wall. While the uterus is firmly contracted, the hand remains quiescent; when it softens, gentle massage of the whole organ is practised. This treatment should be carried on for a minimum of half an hour, unless there are definite signs that the placenta has escaped freely into the vagina—and it is often by no means easy to be sure of this. As soon as the placenta has escaped into the vagina, the patient is asked to strain or cough. Frequently this is enough to cause its delivery. If not, the uterus, when firmly contracted, is used as a piston for pushing the placenta out of the vagina.

If, by the end of half an hour, there is no sign of the placenta having left the uterus and there is no hæmorrhage, time should still be given, but eventually expression may be undertaken. Again, when firmly contracted, the uterus is squeezed from above downwards and then pressed down in the pelvic axis to deliver the placenta. This is effective in all but

pathological cases. Any membrane that tends to be retained should be treated with extreme gentleness. Twisting the whole placenta is useful, combined with an up-and-down movement. If it is still adherent, sterile fingers should be introduced into the vagina up to the level of the cervix, when slight traction is usually sufficient.

After delivery of the placenta, control should still be kept of the uterus for 5–10 minutes, and if there is any tendency to bleeding, pituitrin ($\frac{1}{2}$ –1 c.c.) or ernutin (10 min.) should be given intramuscularly.

Treatment of existing post-partum hæmorrhage.—If there are no obvious tears, immediate efforts should be made to cause the delivery of the placenta. The uterus should be massaged vigorously through the abdominal wall, and, as soon as it is firmly contracted, *expression* should be tried. So long as the organ is well contracted, considerable force may be used to this end.

If expression is unsuccessful, *manual removal* must be resorted to. A sterile condition of the hand and forearm is of first importance in this step, and preparations must be made accordingly as described above. The patient is placed in the lithotomy position with the buttocks over the edge of the bed. One hand is introduced into the vagina; the other controls the uterus through a sterile towel on the abdomen. Examination is made for tears. At the upper limit of the cervix the smooth layer for separation between the uterus and membranes is found. The latter, with the cord, should be held in the palm of the hand. On reaching the placenta, separation should be methodical from side to side and from below upwards. The greatest help is given by the abdominal hand. When the placenta is completely separated it can be manipulated from the parturient canal without removal of the internal hand. The internal surface of the uterus should then again be examined to confirm its smooth condition. Its cavity should be thoroughly douched with a hot (120° F.) lysol douche (0·5 per cent.), the intra-uterine nozzle being guided into position by the hand which remains in the uterus. The hand can then be withdrawn, and after it the nozzle. In the great majority of cases the uterus is now firmly contracted and hæmorrhage ceases.

After its delivery the placenta should always be examined to see that it is complete. If it has been delivered spontaneously, this is usually the case; if manually, it is known that the

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uterus is empty; but when forcible expression has been used a portion may easily be left behind. This is particularly the case when a placenta succenturiata exists, and it may be almost impossible to determine its absence by inspection of the placenta and membranes.

If hæmorrhage begins after delivery of the placenta and the uterus is found large and boggy, *puitrin* ($\frac{1}{2}$ -1 c.c.) should be given intramuscularly and the uterus briskly massaged. If there is no responsive contraction, with the most careful aseptic preparation as for removal of the placenta, the interior of the uterus must be explored for remnants of placenta and membrane, a copious hot douche given, and *binatural compression* of the uterus practised. For this the internal hand, clenched, rests against the anterior wall of the uterus, the knuckles resting in the anterior fornix. The external hand grasps the body of the uterus through the abdominal wall, pressing the posterior wall against the anterior, which is supported from the vagina, so that the whole organ is held firmly between the two hands. Compression may have to be maintained for 15-20 minutes, but is extremely effective. Should it not be successful, the only resource left is that of *packing* the uterus. Antiseptic gauze should be used for this purpose—bismuth gauze or sterile gauze soaked in 0.5-per-cent. lysol. The packing must be methodical and complete, and is best and most aseptically performed by using a speculum and volsella with the patient in the lithotomy position, introducing the gauze with uterine packing forceps. It can, however, also be performed effectively by retaining one hand in the uterus and packing with the uterine forceps under its guidance. The whole uterine and vaginal tract should be firmly packed. An abdominal binder fastened from above downwards is applied to the abdomen, the foot of the bed raised, and restorative treatment given to the patient. This consists mainly in rest, the application of warmth to the body, and replacement of the fluid lost. Normal saline solution may be run into the subcutaneous tissue, a good site being under the breasts, or, if more urgent, into the median basilic or other well-marked vein of the arm. The addition of 5-per-cent. gum acacia is of value in causing the retention of the fluid in the blood-vessels. Blood-transfusion is also of the greatest value, but is not suitable for use in general practice.

When laceration is suspected, careful investigation by inspection and palpation is made. Bleeding-points can sometimes be secured and

ligatured and tears sutured. Sometimes the tear is constantly obscured by blood and, if high and deep, difficult to reach with inadequate help. In these circumstances packing of the whole tract should again be resorted to. The packing may be left *in situ* for 24-48 hours.

General anaesthesia is desirable in all cases in which intra-uterine manipulation is performed, the only exception being where the patient has already lost an extreme amount of blood. It makes treatment easier and safer, and saves the patient shock.

The use of some type of obstetric "crutch" is also invaluable in such cases. Simple types are on the market, consisting of strong webbing with a safety hook at each end and rings into which to lock them. The patient rests immobile in the lithotomy position, and hands are freed for other purposes.

"Late" hæmorrhage occurs at any time in the puerperium after the first twenty-four hours. When serious it is most frequently due to retention in utero of portions of placenta, membrane or firm bloodclot. Sudden dilatation of the uterus may occur, or there may be hæmorrhage from growths, either of the uterine body or of the cervix.

Slighter hæmorrhage is frequently associated with subinvolution or retroflexion, but may also arise from any of the above causes.

If there is no sepsis, and the hæmorrhage is severe, examination should be made and the interior of the uterus explored and treated accordingly.

If there is sepsis, every effort should be made to stay the hæmorrhage without internal treatment; however, if this must be employed to avoid curettage, use ovum forceps or packing forceps clothed with gauze, followed by douching, as a means of clearing away adherent fragments. Under these conditions the uterine wall is very soft and may easily be perforated.

FRANCES M. HUXLEY.

HÆMORRHAGE, SURGICAL.—Hæmorrhage is said to be *primary* if it occurs at the time of the accident or operation, *reactionary* when happening some hours after the severance of the vessel, as shock passes off, and *secondary* if occurring more than twenty-four hours after the injury or operation. Secondary hæmorrhage is nearly always due to sepsis which erodes the vessel and causes sloughing. When the slough separates (from the tenth to the fourteenth day) the bleeding occurs.

HÆMORRHAGE, SURGICAL

Though hæmorrhage, even from arteries, may occasionally stop spontaneously owing to diminished blood-pressure, syncope, or contraction and retraction of the severed vessel, yet this can never be counted upon, and the condition always needs prompt attention.

It is a golden rule in all cases to stop the bleeding by means directed to the bleeding-spot itself. Pressure on or ligature of a vessel at the site of injury is the best treatment.

The various measures adopted to control hæmorrhage are as follows:—

1. **Pressure**, applied by the fingers, by means of a tourniquet, or by the use of gauze plugging, usually serves, at least temporarily, to stop hæmorrhage. Venous bleeding, even in a deep wound, can always be stopped by adequate plugging. *Tourniquets* are of two kinds—either elastic bands, which strangle the limb proximal to the bleeding site (Esmarch's, Samway's); or consisting of a strap and screw-pad which fits closely over the line of the artery (e.g. Petit's). In using the elastic tourniquet it is important to stretch the band well before applying the first turn. In first-aid work a tourniquet can be improvised out of a large handkerchief or cloth-band tied round the limb and tightened by a stick put through the knot and twisted.

Digital pressure may be used to control hæmorrhage from the *subclavian artery*, by directing the fingers downwards and backwards behind the clavicle so as to compress the vessel against the first rib; from the *brachial artery*, by pressing the vessel outwards against the middle of the humeral shaft; from the *femoral artery*, by pressing backwards and slightly inwards at a point midway between the anterior superior spine and the pubic symphysis.

Full *flexion of the knee or elbow*, with a pad applied at the flexure, serves to control bleeding from the leg or forearm respectively.

2. **Position**.—Elevation of a limb will frequently stop venous hæmorrhage, e.g. bleeding from a varicose vein.

3. **Heat**.—Oozing from raw connective-tissue surfaces and from vessels of the pia mater can often be stopped by hot saline lotion at a temperature of 110°–120° F. Sponges wrung out of hot saline will stop bleeding from muscles if pressed firmly against them.

4. **The cautery**.—The galvano-cautery when applied at a dull-red heat will stop bleeding from the mucous membrane of the nose or mouth. The Paquelin or the actual cautery

may be utilized to stop oozing from dense fibrous tissue. Bone should never be cauterized, for local necrosis follows such treatment.

5. **Chemical hæmostatics**.—Adrenalin hydrochloride (1-in-1,000 solution) readily stops oozing from capillaries or small arterioles.

Turpentine, though painful in application, is a useful hæmostatic. Solutions of hamamelis or antipyrin have a similar effect.

Horse-serum (injected subcutaneously in doses of 5–10 c.c.) has a hæmostatic effect.

The effect of small doses of opium in quieting the circulation, and of ergot in causing contraction of arterioles, makes these drugs useful as remote hæmostatics.

6. **Forcepressure**.—Hæmorrhage can always be controlled if it is possible to clamp the vessels with a pressure-forceps. With small vessels, crushing for the period of an operation suffices to obliterate the lumen and stop the hæmorrhage permanently. Large vessels, such as the renal, can be permanently obliterated by applying a strong forcepressure forceps for 48–72 hours.

7. **Torsion** of a vessel after seizing it with forceps is a useful method of stopping bleeding in plastic work.

8. **Ligature** of a vessel is the most certain way of stopping bleeding from it. A ligature should be applied to both ends of a severed vessel or on either side of a lateral opening.

The **best methods of stopping bleeding from different parts** may be briefly summarized thus:

Scalp.—Suturing the wound through the whole thickness of the skin is sufficient. Firm pressure by a capeline bandage may sometimes suffice.

Face.—Either ligature the vessel or insert deep sutures.

Nose.—See EPISTAXIS.

Lip.—Stop the bleeding temporarily by compressing the coronary arteries on both sides of the wound; suture stops it permanently.

Tongue.—If severe, the bleeding may be stopped temporarily by inserting a finger into the mouth as far back as the base of the tongue and pulling the hyoid bone upwards and forwards; the vessel should then be seized with forceps and ligatured. The lingual artery may need to be tied under the hyoglossus.

Tooth socket after extraction of teeth.—The mouth should be rinsed with cold water, or with a dilute solution of alum or tannic acid. Apply a pledget of gauze soaked in solution of adrenalin chloride. Plug with gauze, place

division may allow retraction of the artery and stop the bleeding. A catgut suture passed under the vessel is the best method to adopt.

Stab wounds.—Plugging tightly with gauze will nearly always stop bleeding, but later the wound must be explored after the circulation has been controlled by tourniquet or pressure on the main artery.

Palmar arch.—Temporarily place a firm pad in the palm and bandage the hand over it in the flexed position. At the time that the wound is cleaned and sewn up, the bleeding vessel must be identified and ligatured. Severe secondary hæmorrhage from a septic wound of the palm may rarely necessitate ligature of the brachial.

Varicose veins.—Make the patient lie down. Elevate the leg, and apply a pad and firm bandage over the bleeding-spot.

Tonsils after operation.—Douche the face with ice-cold water. Let the patient gargle with ice-cold water or astringent solution. Apply a gauze pad soaked in 1-in-1,000 adrenalin solution. Catch any spurting vessel with clip forceps and apply a ligature. Finally, the pillars of the tonsils may need to be sutured together under an anæsthetic.

Bone.—Bleeding from a cut surface of bone can be stopped either by crushing the bone with forceps or mallet, or by plugging with Horsley's wax.

Rectum.—Severe bleeding may occur into the rectum without any external escape. Blanching of the skin and lips and a rising pulse-rate are danger signals. If it occurs after an operation for hæmorrhoids an anæsthetic must be given and the bleeding-point caught with forceps and ligatured. If that is impracticable, a small vulcanite rectal tube may be passed into the rectum and strips of gauze packed between the tube and the rectal wall.

Plugging with petticoated tube is permissible in cases of hæmorrhage from inoperable malignant disease.

Bladder.—The viscus may be washed out

to anoxæmia, yawning, restlessness, and other local pain. The temperature is usually sub-normal, but, if the bleeding stops, fever may follow. Operation is usually indicated for abdominal hæmorrhage, but intrapleural bleeding often stops naturally.

Hæmophilia.—In some persons the coagulative power of the blood is deficient and any injured vessel bleeds continuously. No operation should be lightly undertaken in such subjects, not even dental extraction. If operation is necessary, preliminary administration of horse-serum is advisable, and 60 gr. of calcium lactate should be given daily for two or three days beforehand. (See also HÆMOPHILIA.)

After-treatment of hæmorrhage.—After severe hæmorrhage has been stopped there is need—

(a) **To maintain the blood-pressure.**—This is best done by allowing fluids to be drunk freely, or by administering normal saline solution, either subcutaneously, rectally, or intravenously.

(b) **To make new blood.**—In severe cases immediate transfusion of blood from a suitable donor is advisable (see TRANSFUSION). Later, hæmatinic drugs such as iron and arsenic should be given.

ZACHARY COPE.

HÆMORRHOIDS (*syn.* Piles).—A varicose condition of the veins of the anal canal. Hæmorrhoids are termed external when covered by skin, and internal when covered by the mucosa of the anal canal.

Etiology.—Anything which impedes the circulation of the anal region tends to produce piles. A sedentary occupation, constipation, stricture of the rectum, hepatic disease, tumour of the pelvis, or the constant local congestion produced by dysentery, may be predisposing factors. In some patients there appears to be a congenital predisposition.

Pathology.—Internal piles result from dilatation of the terminal tributaries of the superior hæmorrhoidal vein which take origin under mucosa close to the muco-cutaneous junction.

HÆMORRHOIDS

induced by fibrous according to the examination consequent on the use of the finger, or other cause of the disease. In the usual situations for piles, the internal piles are situated in the anterior quadrant, the right posterior quadrant, and the left posterior quadrant of the anal margin.

External piles result from a dilatation of the venules which form a network superficial to the external sphincter. Frequently they give rise to no symptoms till a thrombosis occurs. The redundant folds of skin round the anal margin are sometimes loosely termed external piles. The tag of skin at the base of a fissure is known as the "sentinel pile."

Symptoms of internal piles. These reveal themselves usually by bleeding or prolapse. Pain is sometimes complained of, and occasionally a slight watery discharge may be noticed, especially when the piles are prolapsed. *Bleeding* is the commonest symptom. Usually it is slight in amount at first, but later may be serious. The blood generally comes away after an action of the bowels, but in prolapsed and congested piles a sanious discharge is seen apart from defæcation. Sometimes the constant drain of blood causes the patient to become severely anæmic. *Prolapse* of the piles is often the symptom which causes most discomfort to the patient. Straining at stool causes the thickened congested mucosa to protrude. At first the protruded parts can be returned voluntarily by action of the anal muscles; later the patient needs to return them by digital pressure. The constant dilatation of the sphincters by the protruded parts causes a weakening of the muscle, and thus a greater tendency to prolapse. With a very lax sphincter, prolapse occurs after walking a short distance or after any special effort. *Pain* is sometimes complained of, but is usually no more than a feeling of fullness in the anal canal. Severe pain is generally due to the presence of a fissure.

Complications of internal piles.—Anal fissure and fistula in ano are common accompaniments. Inflammation or thrombosis may occur. A group of prolapsed piles may be tightly gripped by the external sphincter so that the circulation is cut off; strangulation then ensues, the protruded mass swells greatly, becomes very painful, and unless relief is given in time a portion of the piles becomes necrotic.

Diagnosis of internal piles.—The history

of occasional slight bleeding, coming down after defæcation, is suggestive of piles. Examination by finger reveals nothing abnormal, but on the edge of the anal margin the purplish projecting mass may come into view owing to the laxness of the sphincter. Digital examination of the anal canal reveals a slack and toneless sphincter in those cases in which prolapse of the piles has been frequent. The mucous membrane can often be recognized as thickened, and occasionally individual piles may be palpated. By using the proctoscope it is possible to see the piles, but this is not usually necessary.

Differential diagnosis.—Lay people commonly apply the term "piles" to any affection in the anal region. Frequently anal fissure or fistula is so misnamed. The pain of the former and the discharge and sinus in the latter are distinguishing features, though piles may co-exist with either. Prolapse without piles is recognized by the circular ring of fairly normal mucous membrane, which shows no distinct projections, as do prolapsed piles.

Diagnosis is not difficult if care be taken in examination, but if digital rectal examination is not made in every suspected case of piles, the serious mistake will sooner or later be made of missing a carcinoma or stricture of the rectum which has contributed to the formation of the piles.

Treatment of internal piles.—The first indication is to remove any condition which may be causing or aggravating the piles. The bowels must be regulated by aperients and by occasional rectal injection of olive oil if there be constipation. The circulation must be improved by the taking of regulated exercise. Bicycling and horse-riding should be forbidden, or indulged in moderately. Hepatic congestion must receive appropriate treatment, and any pelvic inflammation or tumour dealt with. When, in spite of these measures, symptoms persist, it is necessary to treat by some local application. Ung. gallæ cum opio, ung. hamamelidis, or an ointment containing sulphate of iron may be applied. A suitable application can be made up of bismuth carbonate 20 gr., zinc oxide 20 gr., and vaselin to 1 oz. To this may be added cocaine 2 gr. to the ounce in case of pain, or adrenalin hydrochloride 10 min. to the ounce in case of undue hæmorrhage. Care should be taken that the diet is not of a constipating nature, and alcohol should be indulged in sparingly or, better still, not at all.

HÆMOTHORAX

If these methods of treatment fail to relieve, and in any case in which the bleeding is having a deteriorating effect on the general health, or the piles constantly protrude on the slightest provocation, operation is to be advised. It is contraindicated when cirrhosis of the liver or cardiac disease is present, or when arteriosclerosis has seriously affected the arteries.

Symptoms and diagnosis of external piles.—External piles give rise to no symptoms except slight local irritation, unless a thrombus forms. The thrombosis usually occurs in a local extravasation of blood around the vein. Pain is then felt at the site of the swelling, which forms a bluish prominence at the margin of the anus. The diagnosis is easy, but it is important to remember that patients sometimes complain of piles when the real condition is a condyloma.

Treatment of external piles consists in correcting constipation, which is usually a predisposing cause of the piles, and in keeping the parts very clean.

When the irritation is annoying, the redundant tags of skin should be removed by scissors after injecting a few drops of 1-per-cent. novocain into the skin-fold. Where a local thrombus forms, a few drops of novocain solution should be injected, an incision made, and the clot turned out.

ZACHARY COPE.

HÆMOTHORAX.—A collection of blood in one of the pleural cavities.

Pathology and etiology.—The bleeding takes place either from the systemic circulation (an intercostal or mediastinal vessel) or from the lung.

1. Trauma is the commonest cause—a blow causing a fractured rib, or a penetrating gunshot or stab wound. In the latter case the bleeding may be primary, or may occur later from the rupture of a traumatic aneurysm.

2. Of local diseases, cancer of the thoracic contents is a frequent and tuberculosis a rare cause.

3. Hæmothorax occasionally results from general disease, especially nephritis, cirrhosis of the liver, scurvy, purpura, and hæmophilia.

4. Next to trauma, the commonest cause is the rupture of an aortic aneurysm.

Symptomatology.—The usual signs and symptoms of pleural effusion are present (dullness, diminution or loss of vocal resonance and breath-sounds, compression of the lung, and displacement of the heart), as well as signs of

HAIR-BALL

internal hæmorrhage (pallor, restlessness, thirst, thin rapid pulse, shallow respiration, and lowered blood-pressure). The temperature rises at night to about 100°–102° F.

In cases due to trauma, and especially those resulting from a gunshot wound, pneumothorax and surgical emphysema are often associated with the hæmothorax, obscuring the physical signs. Aspiration will settle the diagnosis.

Cases of "leaking hæmothorax" form an important clinical group in gunshot injuries; fresh or old blood and pleural exudate leak from an open wound of the thorax, greatly increasing the risk of infection, as well as giving rise to much distress from mechanical interference with respiration and the heart's action.

Treatment.—Active treatment of hæmothorax is advisable in all cases resulting from a penetrating wound, and in other cases where great respiratory and cardiac distress is caused by the pressure of the fluid.

In cases of closed hæmothorax due to wounds, aspiration may be carried out within twenty-four hours, and should be repeated every two days if any fresh effusion takes place; by this means infection can be prevented or minimized, and dense pleural adhesions avoided.

In leaking hæmothorax the wound and any fractured ends of ribs should be excised, the pleural cavity irrigated with eusol, and the wound sutured without drainage; the pleura should be kept as dry as possible subsequently by repeated aspiration. Blood-transfusion should be carried out if there has been much bleeding.

If infection of a hæmothorax take place, by a similar operation of opening, washing out, and closing the pleural cavity the inflammatory process can be cut short, and drainage can be avoided in many cases.

C. W. GORDON BRYAN.

HAIR, LOSS OF (see ALOPECIA).

HAIR-BALL.—Hair-chewing and -swallowing may lead to the formation of a felted mass of hair in the stomach. The mass may tail off into the œsophagus above or through the pylorus below. Children, especially girls, and neurotic or demented women are most prone to this habit. The patient is usually seen for symptoms of abdominal discomfort, and examination reveals a hard tumour in the position of the stomach; it may impart a curious constant sensation to the palpating hand. It may

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freely together with the stomach, and may be moved about in the organ, a sensation of "ballottement" being obtained. The diagnosis is made, in the presence of this tumour, by the history, the appearance of broken-off hair or bald patches, and the occurrence of wisps of hair in the motions. The passage of a tube is confirmatory, for it cannot pass through the cardia to an appreciable extent, nor is it possible to pour more than a little fluid into the stomach. Hair may be found in the fluid returned. Radiographic appearances are very characteristic after an opaque meal. There is at first an opaque cap at the cardia surmounting the less complete and defined opacity of the hair. Later the meal, permeating between the hair-ball and the stomach-wall, portrays the outline of the stomach distinctly. Palpation under the fluorescent screen may assist the diagnosis materially. A similar condition has resulted from the chewing of fluff from blankets. The only effective treatment is by operation, which is usually successful. More rarely, masses of hair may be found in the intestine, where they may cause obstruction.

FREDERICK LANGMEAD.

HALLUCINATIONS. — An hallucination may be defined as a sense-perception experienced in the absence of any objective stimulus. Thus, if a patient hears a voice speaking to him when actually complete silence exists, he is said to experience an auditory hallucination. An hallucination is technically distinguished from an illusion by the fact that in the latter an actual objective stimulus exists but is erroneously perceived. An illusion is present, for example, when a delirious patient perceives the pattern on the wall-paper as crawling insects. The distinction, however, has but little practical importance, and in all probability the mechanism of production is identical in the two cases.

Hallucinations are a prominent feature in various types of insanity, and have been estimated to occur in from 50 to 70 per cent. of all cases. It must be clearly understood, however, that their existence does not necessarily indicate that a psychosis is present, and that they occur in other conditions also. They are met with, for example, normally in dreams, and in the transitional stage between sleeping and waking (hypnagogic hallucinations). They occur also in hysteria, and can be easily produced artificially in hypnosis.

Hallucinations may concern any of the senses and are termed auditory, visual, tactile, and so forth, according to the particular sense affected. The first two are much the most common. In addition to those relating to the five chief senses, hallucinations of temperature and pain may occur, and there are also psychomotor hallucinations in which the patient experiences a feeling of movement in some part of the body without any actual movement occurring.

In determining the significance of the existence of hallucinations in a given case it is of the first importance to ascertain the light in which they are regarded by the patient. He may himself recognize that the phenomenon is subjective, and that the object which he perceives has no real existence. This recognition is always present, either at the time of or immediately after the experience, when hallucinations or illusions occur in normal persons or in psychoneurotics. It is practically always absent when hallucinations occur in the psychoses (insanities), and it therefore furnishes a rough practical guide as to whether the condition under examination is to be classed with the psychoses or not. Hallucinations whose subjective character is recognized by the patient are sometimes termed pseudo-hallucinations.

Hallucinations are common in the deliria, exhaustion psychoses, alcoholic psychoses, paranoid psychoses, and in dementia præcox. They are relatively uncommon in the manic-depressive group of insanities. Hallucinations are frequently associated with delusions, and are sometimes said to cause the latter. It is probable, however, that this is incorrect, and that the two phenomena are properly to be regarded as concomitant manifestations of the same morbid process.

The precise mechanism of production of hallucinations and illusions is the subject of considerable dispute, but it seems clear that the process is essentially central and not peripheral. A stimulus or other change may occur in the peripheral sense organ, but its presence is not necessary, and in any case it probably only serves as a point of attachment upon which the centrally produced hallucination is projected. For this reason hallucinations and illusions are not to be regarded as essentially distinct from one another, but merely as varieties or, rather, degrees of one and the same process.

BERNARD HART.

HALLUX VALGUS

HALLUX VALGUS.—A condition of deviation of the big toe outwards towards the other toes; it is usually accompanied by flat-foot and frequently by hammer-toe. The common cause is badly fitting footwear which cramps the toes; this is aggravated by walking or standing with the toes turned out, for thereby excessive pressure falls on the big toe and inner border of the foot. In a well-marked case the big toe is bent out at the metatarso-phalangeal joint sometimes to an angle of 45° , so that it encroaches on the space usually occupied by the second toe, which is thus thrust aside and may rest upon the upper surface of the misplaced hallux. The head of the first metatarsal bone is very prominent, and new bone is formed as the result of irritation of the exposed area. An adventitious bursa (bunion) frequently forms over the prominence. The disabilities caused by hallux valgus are pain and discomfort in walking, with the additional inconvenience of the bunion, which often becomes inflamed.

Treatment.—It is very important to treat any accompanying flat-foot by the usual measures (correct boots, tip-toe exercises, etc.). In slight cases of hallux valgus it suffices to instruct the patient to acquire boots of natural shape with a straight inner border and square toe-portion allowing plenty of room, and to advise him always to walk with the toes pointing straight forwards. Further relief is obtained by thickening the inner border of the boot by about $\frac{1}{2}$ in.; it is well also to have a separate compartment in the sock for the big toe.

In slightly more pronounced cases mechanical means are needed in addition. During the night the patient should wear a narrow splint along the inner border of the foot; if the splint be bound firmly to the big toe, and then approximated and fixed to the foot, the deformity will thereby be corrected. During the day the toe can be kept in correct position by fixing a piece of strapping round the big toe and sticking the long end of the loop along the inner border of the foot as far as the heel; or a toe-post may be placed in the boot.

In severe cases, especially those with a troublesome bunion, operation is advisable. Excision of the head of the metatarsal bone is the best procedure. A longitudinal or curved incision is made over the head of the bone, which is cleared by using knife and raspator, and removed by chisel or bone-forceps. The tendon of the extensor proprius hallucis may

HAMMER-TOE

be transplanted to the inner side of the big toe.

When a bunion is present, Mayo's operation is to be recommended. A small semicircular flap of skin with horizontal base is dissected up, then the tissues between skin and bone, including the bunion, are dissected up in an elongated flap with vertical base just opposite the joint. The head of the bone is removed and the bunion inserted into the joint; it serves as a new synovial cavity. The patient may be allowed to walk in a few days, though active exercises should not be allowed till healing is sound.

ZACHARY COPE.

HAMMER-TOE.—A condition affecting almost exclusively the second toe, whereby its first phalanx is hyperextended, the second flexed, and the terminal phalanx either extended or flexed. It may occur in several members of one family, and may be of congenital origin. It is not open to doubt, however, that the majority of cases are due to the wearing of boots which tend to alter the natural shape of the foot. When the condition has lasted some time there are shortening of the anterior and lateral ligaments of the first interphalangeal joint, and contraction of the skin, subcutaneous tissues, and flexor tendons of the toe; the base of the second phalanx is then displaced forwards and the head of the first phalanx exposed. Over this prominence usually develop a painful callosity and an adventitious bursa. The extremity of the toe may be flattened out.

The consequences of hammer-toe are pain and discomfort in walking, often aggravated by the presence either of soft or of hard corns.

Treatment.—From the point of view of treatment three degrees of hammer-toe may be considered. (a) When the deformity can be corrected completely by manipulation and there is no contraction of the ligaments, it is sufficient to order daily manipulation and to advise the wearing during the night of a narrow splint along the plantar surface of the toe extending on to the foot. The boots must fit well and, while they are on, the toe may be kept in place by a piece of strapping (the sticky surface upwards) passed over the prominence of the affected toe and under the adjacent toes. (b) For the second degree, in which there is shortening of the ligaments, a subcutaneous tenotomy of the contracted parts is needed in addition to the above treatment.

HARE-LIP

This may be carried out, if necessary, under local analgesia. (c) The third degree comprises the severe cases. Here it is essential to excise the head of the first phalanx and remove the overlying callosity. An oval dorsal incision is made round the callosity, the head of the bone exposed and removed with bone forceps, care being taken of the extensor tendon. A plantar toe-splint is applied. After about ten days the splint should be discarded and normal walking encouraged.

Correction of a hammer-toe is hardly ever necessary, and if carried out may lead to the formation of any coexisting hallux valgus.

ZACHARY COPE.

HANGING (see ASPHYXIA).

HANOT'S CIRRHOSIS (see LIVER, (CIRRHOSIS OF)).

HARE-LIP.—Hare-lip may be single, in which case the defect is present on one side only, or double, when it is present on both sides of the lip. Either variety may be complete or incomplete. In the former the gap extends into the nostril; in the latter it involves the lip to a greater or less extent, varying from a mere notch in the mucous border, to one which is only separated from the flattened nostril by a thin bridge of tissue. In the complete form the nostril is always deformed, being flattened, thinner, and broader than the normal. In single hare-lip the premaxilla may have failed to unite with the maxilla on the affected side. In these cases the premaxilla is often placed obliquely and tilted forwards, and the palate is completely cleft. In double or bilateral hare-lip the premaxilla is attached to the end of the nasal septum, where, covered by the skin of the probium, it forms an unsightly projection; a complete cleft of the hard and soft palates is usually though not always present.

In many cases of incomplete hare-lip the tissues above the cleft are thinner than normal. In complete clefts the sides of the gap are lined with mucous membrane continuous with that of the lip and extending into the nostril; or the mucous membrane may tail off, a narrow strip of skin completing the margins of the cleft on each side at the upper end. These points are important because, in operating, the incisions must be made with a view to removing all such portions of skin or mucous membrane inside the nostrils. The lip itself may be thin,

and be loosely or closely attached to the maxilla; it may be thicker on one side of the cleft than on the other.

As the child is generally unable to suck, artificial feeding must be carried out from the time of birth (see CLEFT PALATE). The operation to remedy the condition should be done as soon as possible, which is generally about the third or fourth week; if the child is particularly robust and healthy there is no reason why it should not be done at an earlier date. On the other hand, in a weak and feeble infant it is advisable to postpone it. The most important points in performing the operation are—(1) to make the lip as broad as possible from above downwards; (2) to make the nostril as round as possible; (3) to ensure accurate apposition of the line of junction of the mucous membrane of the lip ("the red portion") and the skin ("the white portion") on each side of the defect.

After-treatment.—It is often possible to do without dressings after the operation. After drying and removing all blood from the lip, collodion may be painted over the line of suture. If there is much tension on the stitches, a piece of strapping, cut to a dumb-bell shape, may be applied to each cheek, and gauze may be placed under the strapping and over the lip. This will require changing frequently, as it soon becomes sodden.

The wound must be kept as clean as possible and the nostrils free from dried mucus. Small drosses of wool held in forceps and soaked in sodium bicarbonate solution (10 gr. to 1 oz.) are most useful for this purpose. Mild antiseptic lotions may be used to bathe the operation area. The infant should not be allowed to cry more than is absolutely unavoidable. Hunger is a common cause of crying, and therefore feeding should be started as soon as possible and continued at regular intervals. By careful nursing and, possibly, the administration of a small dose of bromide of potassium, crying in most cases can be reduced to a minimum.

For a few hours after the operation there may be some obstruction to the breathing. This is easily remedied by placing the thumb on the lower lip and everting it, and at the same time slightly depressing the lower jaw.

No definite date can be given on which the stitches should be removed. As a rule they should not all be taken out at the same time. Careful inspection will show when any of them can come away; some may be taken

out as early as the fifth day. That in the nostril should usually remain longest, for too early removal of this stitch is often followed by widening of the nostril.

The question whether the hare lip should be operated on before the palate has frequently been discussed. In my opinion it is best to operate on the lip as soon as possible. I have not found that closure of the defect in the lip interferes with the subsequent operation on the palate.

Secondary operations are not infrequently required to repair defective primary operations. The common defects of the latter are—(1) faulty union throughout the whole width of the lip, (2) deformity in the region of the nostril or at the free margin of the lip, and (3) insufficient paring of the edges.

T P LEGG

HARVEST-BUG RASH (*syn* Leptus Autumnalis).—A disease characterized by an eruption about the ankles, legs and waist, occurring chiefly in July and August, among those who come into contact with field grass and bushes, and due to a tiny parasite. The lesions, which are intensely itchy, consist of bright-red papules which may become urticated. Treatment consists in the application of weak parasiticides, such as sulphur, balsam of peru or carbolic acid. It is stated thatunction with tincture of benzoin acts as a deterrent.

H MACCORMAC

HAY FEVER.—A catarrh of eyes and nose due to a soluble toxin present in pollen of flowering grasses and other plants, and prevalent during the seasons when such plants are in flower. In Europe it is chiefly due to the grasses, and is therefore most prevalent in May and June, but in America the pollen of golden rod and ragweed are more important, these flower later in the year, and the disease in that country is sometimes called autumn catarrh.

Etiology.—Some persons are peculiarly susceptible to the pollen toxin. The susceptibility is considered to be due, in part at any rate, to the nervous system, and it is said to be more prevalent among the upper classes of society in general, and among the sensitive and intellectual in particular. The susceptibility may be acquired by long exposure. It is probable that gross defects of the respiratory tract, such as hypertrophic conditions of the nasal mucous membrane, or bony obstructions, also predispose to or aggravate the condition

among the susceptible. The exciting cause is quite definitely proved to be a toxalbumin present in the pollen.

Pathology.—The morbid anatomy consists in intense congestion and catarrh of the conjunctivæ, nasal passages, and adjacent parts.

Symptomatology.—About the time when the grass pollen is shed, which is in May, the symptoms are liable to begin, but at this time are likely to be local and confined to persons living in the neighbourhood of hayfields. The pollen is produced in immense quantities, and in the following weeks is widely distributed in the atmosphere, so that the disease is most rife and most severe in June. The severity of the attacks is much greater in dry weather, since in wet seasons the pollen is far less carried about. The initial symptoms are usually an itching of the eyes and a sensation as if they contained grit, with itching of the nose followed by attacks of sneezing. When the attack is severe, these symptoms are much increased, there are an intense hyperæmia of all the parts, profuse excretion, sometimes chemosis of the conjunctiva, which in severe cases is even shed, intolerable itching of the palate and fauces, and, in some persons, definite bronchial asthma. The sneezing may be repeated hundreds of times. The consequences are very serious exhaustion, irritability, and depression, some patients being quite incapacitated for work.

Diagnosis can be made with certainty by installation of pollen extract into the conjunctival sac, this produces redness of the caruncle when used in very dilute solution upon patients who are susceptible to hay fever, but will produce no effect when strongly concentrated in insusceptible persons.

Prognosis.—In cases untreated by specific methods the condition is very intractable, the severity varies with the season, but the affection almost invariably recurs, cases of spontaneous recovery being very rare. Great improvements have, however, been effected in recent years by specific treatment.

Treatment.—Specific treatment may aim either at active or at passive immunity. As to the latter, Dunbar's serum is obtainable under the name of "pollantin" either in solid or in liquid form. It is highly concentrated, and a small drop or a small pinch is put into the conjunctival sac as a prophylactic measure before exposure to the pollen. Dunbar claims very good results, distinct benefit having been received in about 700 out of 1,240 patients.

subject to the limitations and short duration inherent in this method.

Active immunization was tried first by the late Leonard Noon, and has been followed up by Freeman in Sir Almroth Wright's laboratory. By this method pollen extract is injected subcutaneously into the patients themselves, who are thus stimulated to form their own antibodies. The diagnosis must first be made by the ophthalmic test. The unit of pollen extract adopted is the amount obtained from 1000 mg. of pollen; various strengths are made up, and drops of successive strengths are put into the conjunctiva. The initial subcutaneous dose is $\frac{1}{2}$ c.c. of an extract of the strength causing a reaction.

As a prophylactic measure, the patient should be reinoculated every week or ten days, each dose being half as much again as the previous one, and progress being checked occasionally by the ophthalmic reaction. It is best to begin prophylactic treatment about three months before the hay season begins. Treatment after hay fever has begun frequently gives satisfactory results, but prophylaxis is obviously preferable.

In Freeman's opinion, immunity lasts at least a year after treatment has been discontinued.

Non-specific treatment.—Persons of means and leisure can avoid hay fever by going to sea or to such places as the Outer Hebrides during the season. Gross abnormalities of the nose should receive surgical treatment. For local treatment, oily sprays are perhaps the most effective and form some protection to the mucous membranes, and antiseptic sprays are sometimes beneficial. Burney Yeo recommended resorcin $1\frac{1}{2}$ gr., sodium chloride 4 gr., acetic acid 2 min., water to 1 oz.

D. W. CARMALT-JONES.

HEADACHE.—Headache may be due to so many causes, and is associated with so many disordered states, that it can be regarded only as a symptom. Consequently, the main aim in treating every case must be to determine and then remove the cause to which it is due.

In order to investigate patients properly it is necessary to have a classification of headaches according to their cause, for without this we cannot hope to explore fully the possible factors. But the formulation of a classification presents many difficulties, owing particularly to the fact that we are still ignorant

of the mode in which headaches arise. The brain itself is certainly insensitive to direct stimulation; no matter what form of irritation is employed, no sensation referred to the head is evoked. When the scalp, skull, and membranes are made insensitive by local anæsthesia, it can be handled, compressed, and incised without producing any sensation similar or analogous to headache. The meninges—or at least the dura mater—are, on the other hand, sensitive; the arachnoid contains no nerves and the pia mater receives only sympathetic twigs for its blood-vessels, but the dura is supplied with a large number of sensory fibres which leave the three divisions of the trigeminal nerve distally to the Gasserian ganglion and pass to this membrane as recurrent meningeal branches. The most important of these are the anterior and posterior ethmoidal and the tentorial nerves from the ophthalmic division, the middle meningeal from the second division, and the recurrent branch from the mandibular division that re-enters the skull through the foramen spinosum. A recurrent branch from each vagus also contributes to the supply of the dura of the posterior fossa.

There can be no doubt that these dural branches are concerned in the production of headache; we have direct evidence of it in the fact that, after extirpation of the Gasserian ganglion, headaches rarely or never occur on the same side of the head, and I have seen patients previously subject to migraine who ceased to suffer with unilateral headache after the ganglion had been injured by an attack of herpes. It might be assumed that not all headaches are due to irritation of the dural nerves, but it is obvious that pain can be felt only through the mediation of nerves, and these are the only sensory filaments that are distributed to the intracranial cavity. The dural nerves may be stimulated in many different ways. In the first place, by a rise of pressure within the skull, such as is produced by cerebral tumours and abscesses, meningitis, and congestion of the cerebral vessels. In the second place, these nerves may be irritated by poisons and toxins circulating in the blood, as alcohol, nicotine, the toxins of the infectious fevers and of nephritis. Thirdly, headache may be a referred pain. Ross, Mackenzie, and Head have shown that in disease of any visceral organ pain may be referred to a corresponding area on the surface of the body by the disease causing a state of abnormal excitability of that portion of the grey matter

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of the cord in which the visceral afferent fibres terminate. In the same way, disease in the distribution of the trigeminal nerve may excite pain that is referred to the head. The headache of *nasal disease* and that associated with disease of the nose and its accessory sinuses are common instances of this condition. Head has also shown that local headaches may be a result of pain referred from one of the thoracic or abdominal viscera; this is due to the close central connexion of the afferent fibres of the vagus nerve with the central root of the trigeminus. It is possible that the dural nerves may be irritated in other ways, but these three are certainly the most important.

Clinical examination.—To obtain a complete history of the headache, knowledge on the following points is essential: (1) Its *position*, whether general or circumscribed; the position of greatest intensity, and whether it is superficial or deep in the head. (2) The *time of day* in which it occurs or becomes more severe, whether it is constant or variable, and if it is liable to occur at more or less regular intervals. (3) The *nature* of the pain; it is important to ascertain this as accurately as possible from the patient, but a satisfactory description is rarely obtainable, since our language does not contain sufficiently precise terms for such pathological sensations. We can at least learn whether it is a feeling of pressure or a deep boring pain, a constant dull aching or a throbbing pain, an intermittent shooting pain or a feeling of bursting of the head. (4) *Exciting or aggravating causes*: what it is that starts the headache, and whether it becomes worse on lying down or walking about, when quiet or under excitement, and if food, or exposure to cold, worry, or work, has any influence on it. (5) *Conditions associated with the headache* should be carefully investigated, as they frequently throw light on its origin. The history should always include reference to the presence of vomiting or gastric disturbances, or vaso-motor and cardiac symptoms, to the mental state during the attack, if the scalp becomes tender during or after the headache, and whether it is accompanied by vertigo or ocular symptoms. (6) *Heredity* should be inquired into; whether ascendants or collaterals are subject to headaches, and if these are of the same nature.

The following classification has been drawn up with a view to practical use, but it must be admitted that it may not be possible to include all headaches in the categories suggested here.

Migraine.—Migraine is dealt with under its own name, but it must be insisted upon here that this is perhaps the most common cause of severe headache. Despite the writings of Liveing and others, it is not yet generally recognized that the vomiting, visual phenomena, and paræsthesiæ which belong to characteristic migraine are present in only a relatively small proportion of cases; teichopsia, for instance, probably occurs in not more than a fifth of all cases. The periodicity of the attacks, their tendency to occur in women at the menstrual periods, their duration from early life, and the history of similar attacks in near relatives are important diagnostic points. Most of the "familial" and "constitutional" headaches are migrainous.

Headache from raised intracranial pressure.—Any condition that raises intracranial pressure may produce headache by compressing the dural nerves. The most important causes are tumours and abscesses of the brain, meningitis, and cerebral syphilis. Headache is also not infrequently due to high blood-pressure, especially when associated with arterio-sclerosis. That which frequently follows cerebral concussion and other cranial injuries is probably of this nature too. It is often an early symptom in general paralysis.

The headache which is an almost constant symptom of *cerebral tumour* is generally described as a dull, deep-seated pain; sometimes it is spoken of as an intense throbbing pain, or a feeling that the skull would burst open from its severity; but often the patient asserts that it is indescribable, something entirely beyond ordinary experience. As a rule it is general, but is often most severe in the frontal region or behind the eyes; occasionally it is worse in the occipital region, especially when the tumour lies in the posterior fossa, and then the pain frequently radiates down the back of the neck. It is, as a rule, local only when the tumour involves the dura mater or the bone, and it may then be accompanied by local tenderness of the skull to pressure or percussion. The pain is usually more or less constant, though severe exacerbations that may last for hours or sometimes even days occur; but many patients are free for considerable periods. An important feature is that it is usually most severe at night. Vomiting is generally associated with the severer bouts of pain, and not uncommonly, if the intracranial pressure be very high, the patient may fall into a stuporous state in which death

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occur suddenly. The presence of papilloedema and symptoms of progressive cerebral disease are important signs in diagnosis. Too much emphasis cannot be laid on the necessity of a routine examination of the optic discs in all cases of severe and persistent headache.

In *acute meningitis* the headache is usually general, but more intense in the occipital region. It is accompanied by stiffness of the neck, and is aggravated by every movement of the head on the vertebral column and by pressure on the suboccipital region, as these increase intracranial tension by compressing the distended posterior subarachnoid cistern.

Headache due to *syphilitic involvement of the brain* may either occur soon after infection, or be associated with the later manifestations of the disease. In early syphilis the pain is more or less constant and spreads from the occiput to the vertex or forehead. It is often particularly severe when the patient lies down or lowers his head. There are usually no local or general signs of cerebral disease associated with it. It may be a result of the toxins reaching the brain as the infection becomes generalized, but is more probably caused by the early vascular and meningeal changes that often occur at this stage. Headaches in the later stages of syphilis may be due to meningitis, gummatous infiltrations, or cerebral vascular disease.

Cerebral vascular disease and arterial hypertension are not uncommonly causes of headache. The arterio-sclerotic variety occurs, as a rule, in the fifth and sixth decades of life, in men more frequently than in women, and usually in those who have led active and vigorous lives. It is generally associated with other more or less characteristic symptoms, as premature senility, mental deterioration and vertigo, and frequently insomnia, or disturbed sleep at night with drowsiness during the day. The headache is generally constant but never severe, it is increased by mental and physical exertion, by worry, and by bad ventilation. Cerebral arterio-sclerosis is not difficult to diagnose when the arterial disease is general, or when it occurs in advanced years, but it may be overlooked when it is the cerebral vessels that are chiefly diseased. The association of symptoms is, however, very characteristic, and valuable confirmatory evidence may be obtained by the ophthalmoscope, as the state of the retinal arteries is the best guide we possess to that of the vessels of the brain.

There is a form of headache that is probably

due to a *temporary enlargement of the pituitary gland*, either as the result of a stimulus or of a physiological demand made on it. It is most common in adolescence and in early adult life. The pain is usually periodic and may last from an hour to a few days; in women it is more liable to occur at the menses. It is deeply seated behind the eyes or in the temples, and may, when severe, be accompanied by vomiting. An enlargement of the sella turcica can sometimes be demonstrated by radiographic examination, and there are occasionally signs of disturbances of pituitary function. The administration of pituitary-gland preparations will usually relieve these headaches, unless they are due to tumour formations.

The **treatment of headache due to raised intracranial pressure** must be directed to the removal or alleviation of the cause. When a tumour or abscess is present, the only rational proceeding is its removal, provided that it is localizable and accessible. If this is not possible the pain may be relieved by a decompression operation, preferably in the subtemporal region, but decompression is justifiable only when the primary disease cannot be eradicated. Various drugs, such as bromides and the coal-tar preparations, often give temporary relief. In meningitis lumbar puncture, repeated daily if necessary, is often successful in controlling the pain. Cerebral syphilis demands vigorous antisiphilitic treatment, preferably by mercurial inunctions. Traumatic headaches require prolonged rest in bed.

In cerebral arterio-sclerosis the patient's mode of life must be carefully regulated; diet should be light, constipation must be avoided, and alcohol limited or prohibited. The patient is almost invariably better in the open air and country life is consequently advisable. A moderate amount of regulated exercise is usually advantageous, but vigorous exertion must be forbidden. Iodides combined with bromides are usually the most effective drugs; nitrites in alkaline mixtures are also frequently of value.

Vaso-motor headaches.—Vaso-motor disturbances are frequently assumed to be causes of headache, but their importance is uncertain. There is certain evidence that migraine is due to a periodic vaso-motor upset, and the headaches associated with disorders of the ductless glands and with certain physiological processes, such as menstruation, may be of this nature. That which follows prolonged physical and mental work, and the headache that succeeds

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epileptic seizures, are probably due to disturbances of the cerebral circulation. The most common cause of headache of this nature is cough, which raises pressure in the venous circulation and thus produces venous congestion of the brain. The distressing headaches that sometimes occur in phthisis, bronchitis, asthma, and pertussis are directly due to this cause. A rare variety of headache of sudden onset and extreme severity that occasionally occurs in persons subject to angio-neurotic oedema is probably due to patches of oedematous infiltration of the brain or meninges.

The treatment of headaches due to circulatory disturbances is usually easy and effective. Cough must be checked by appropriate means, and over-exertion must be avoided where this is the cause. When true vaso-motor disturbances are responsible, preparations of the ductless glands should be tried. Belladonna combined with bromide is also of value.

Toxic headaches may be due either to exogenous poisons, as alcohol, nicotine, carbon monoxide, and lead, or to toxins manufactured within the body, as those developed in the specific fevers, pneumonia, and influenza, and those produced by gastro-intestinal disorders and in nephritis. The headaches that are so common in chlorosis are probably of toxic origin too. In fact, most poisons that circulate in the blood cause headache, probably by their irritant action on the meningeal nerves.

The character and the situation of the pain depend on the nature and severity of the intoxication. It is usually vertical or diffuse and of an intense bursting or boring character in acute infections, and a more or less constant dull aching in chronic poisoning.

In acute intoxications treatment can be rarely more than symptomatic. Rest, preferably in a dark room, with cold applications to the head and forehead, and the administration of such drugs as antipyrin, phenacetin, caffeine, and aspirin, generally give some relief. In the more chronic cases our duty is to remove the cause. The importance of gastro-intestinal intoxication must never be forgotten; occasionally a purgative alone is sufficient, but more radical and systematic treatment is generally required. In chronic constipation purgation may aggravate the headache, as by stirring up the faecal contents and making them more fluid it may facilitate the absorption of poisons; regulation of the diet and the use of liquid paraffin are usually more effective remedies.

Referred headaches.—The eyes are certainly the most important source of this form of headache. The intense pain referred to the frontal and temporal regions which is often associated with acute glaucoma and iritis is well recognized. Eyestrain also may be a cause, but its importance is often exaggerated; it is generally associated with hypermetropia or hypermetropic astigmatism, but it may occur when refraction is normal, especially in feeble and debilitated subjects, if the eyes are strained, as by a long visit to a picture gallery or cinema. The pain is usually bilateral and either frontal or temporal; a vertical headache is rarely due to an error of refraction alone. As it usually develops only after the eyes have been used for a time it is, in contrast with migrainous and other headaches, seldom present or severe in the morning.

The ears are a rarer source of referred headache, but when the middle ear is involved by an acute inflammation the pain almost always spreads to the parietal and vertical regions of the head, and is often accompanied by tenderness of the scalp. Then the serious question may arise whether the headache is due to ear disease alone, or to intracranial complications, as an extradural or cerebral abscess, or a serous meningitis. The diagnosis must depend on the presence or absence of local cerebral symptoms and of signs of raised intracranial pressure. Chronic ear diseases also, as otitis media, mastoiditis, and cholesteatomata, may produce temporal and vertical headaches.

Referred headaches of nasal origin are more commonly due to hypertrophy of the turbinates or deviation of the septum, so that they press against one another. But headaches may also result from the absorption of toxic products when drainage is imperfect, and from inflammatory diseases of the nasal sinuses. It is generally a fixed pain with periodic exacerbations, in the frontal and temporal regions, but it is occasionally occipital. Adenoids are a common cause of headache in early life and adolescence.

Diseases of the teeth and deep-seated abscesses in the jaw may produce pain referred to the temporal fossae which the patient may describe as headache.

The thoracic and abdominal viscera may, as already mentioned, sources of pain referred by way of the vagus to the head, but of reference is a relatively rare and unimportant cause of headache. Its direct cause may be the constitutional or circulatory disturbance.

HEADACHE

intoxications produced by the primary disease in these viscera. Gastric headaches are generally localized in the parietal region, cardiac headaches in the frontal and vertical regions, and those due to pelvic disease in the occiput.

The only rational and effective treatment is the removal of the source of irritation. Headache can be often relieved immediately by the correction of an error of refraction, or by the removal of an enlarged turbinate bone. Drugs can give only temporary relief.

Headache due to disease of the scalp and skull.—A true neuralgia is rarely confused with headache. This mistake is most liable to happen when the pain is due to a local disease that involves the nerves, as caries or malignant disease of the base of the skull or of the cervical spine. Severe radiating pains in the occipital region, which are not uncommon in tabes, are often described by the patient as headache. Periostitis and osteitis of the skull, especially when due to syphilis, produce headache as well as local pain.

Nodular or rheumatic headache.—Certain Swedish and German authors regard this as a very common form of headache; it is certainly not uncommon, though less frequent, in England at least, than they assume it to be. Women above middle age are most often affected. The pain starts in the occipital region and radiates over the head, and sometimes into the neck too; it is intermittent, but the patient is rarely quite free during the intervals. It is increased by exposure to cold and chills, and by tension on the posterior cervical muscles, as when the head is allowed to drop forwards in reading or writing. When the back of the head and neck are carefully palpated, small nodules, variable in size and very tender to pressure, can be felt in the subcutaneous tissues, especially at the insertions of the muscles into the skull. The nature of

these nodules has not yet been satisfactorily determined; they are usually regarded as a result of a local fibrositis of rheumatic origin.

The treatment of nodular headache is easy and generally very satisfactory. Massage of the neck and back of the head is essential; it should commence with very gentle rubbing of the nodules, and become firmer and deeper as the tenderness disappears. At first it aggravates the pain, but if persisted in begins to bring relief within ten days or a fortnight. Hot fomentations, or preferably large poultices, should also be applied to the back of the head and neck two or three times a day.

Neurasthenic headaches can be often recognized by the description the patient gives of them and by the circumstances that excite and aggravate them. The neurasthenic headache may be general, but it is more frequently vertical or frontal. It is usually described as a feeling of weight on or constriction of the head, or as a sense of emptiness or vacuity within the skull. It is often, like the other symptoms of neurasthenia, worse in the morning, and is caused by any emotional disturbance, worry, or excitement. It disappears if the patient's mind is taken up by anything else. Finally, it is usually accompanied by a tenderness of the scalp to light pressure or to gentle traction on the hair. These features make it generally possible to distinguish neurasthenic headaches from headaches of organic origin, but it must not be forgotten that in neurasthenia, "the neurosis of exaggeration," there may be an organic basis for the pain of which the patient complains, though not enough to account for its apparent severity.

Treatment should be directed to the disease rather than to the headache. Analgesic drugs should be avoided.

GORDON HOLMES.

